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INFANT BIRTHWEIGHT, GESTATIONAL AGE AND MORTALITY BY RACE/ETHNICITY: A NON-PARAMETRIC REGRESSION APPROACH TO BIRTHWEIGHT OPTIMA IDENTIFICATION

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INFANT BIRTHWEIGHT, GESTATIONAL AGE AND MORTALITY BY RACE/ETHNICITY: A NON-PARAMETRIC REGRESSION APPROACH TO BIRTHWEIGHT OPTIMA IDENTIFICATION

by

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In order to better understand the statistical relationship between measures of birthweight and gestational age and their effects on infant mortality, national vital statistics data was examined using non-parametric regression techniques (GAM) that allow for a sophisticated and detailed analysis of infant mortality models. These models allow for various non-linear effects of birthweight and gestational age on infant mortality to be quantified based upon extant methodologies (Solis, Pullum and Frisbie, 2000). Utilizing over-time, race/ethnic- and sex-specific approaches, the identification of "zones" of optimal birth outcomes based upon infant mortality probabilities is successfully accomplished. This process results from the creation of a rigorous cross-classification of GAM-supplied birthweight and gestational age parameters. From these results, I find

that Non-Hispanic Black infants still exhibit an infant mortality disadvantage relative to Non-Hispanic Whites and Mexican American infants. For the four birth outcome parameters and their interactions, I find evidence of infant mortality disadvantage for infants that are early *or* late as well as small *or* heavy relative to their race/ethnic-specific, birthweight-adjusted optima.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER 1: AIMS, BACKGROUND AND SIGNIFICANCE

1.1 General Introduction and Overview

Recent studies have reported an increasing relative gap between non-Hispanic Black and non-Hispanic White infant mortality in the U.S. even as both groups have experienced large reductions in infant mortality over the last 30 years (Alexander et al. 1999; Guyer et al. 1998; Wise 2003). At present, non-Hispanic Black ("Black") infants are twice as likely to die in their first year of life relative to non-Hispanic White ("White") infants (Mathews, Menacker & MacDorman 2004). Attempts to explain these disparities via known risk factors and proximate determinants have remained tentative at best as their effort toward statistically reducing race/ethnic infant mortality disparities have only been partially successful. Research on race/ethnic groups other than Whites and Blacks has been slow in coming and has only recently become an important focus of most infant mortality studies since the early 1990s. Not coincidently, recent research has also documented changes of historic nature in the structure of infant mortality patterns in the US over the last two decades (Gortmaker and Wise 1997; Wise 2003). This dissertation's general focus is the advancement of our knowledge of differential race/ethnic infant mortality risks and their associated changes throughout the 1990s.

It has been well documented over seven decades that the two most important risk factors related to infant mortality, irrespective of race/ethnic background, are infant birthweight and gestational age (Buekens et al. 2000; Cramer 1987; Paneth 1995; Wilcox and Skjœrven 1992). These two outcomes have been strongly linked to both overall infant mortality risk (IMR) and differential IMRs between subpopulations. Although the etiological pathways are numerous, much research has been devoted to track the associated causes of death linked to early birth and low weight (cf Callaghan et al. 2006; Sowards 1999). Of the better known etiological and physiological causes of death, many are directly or indirectly related to these two birth outcomes. These include respiratory distress syndrome, bacterial sepsis of the newborn, neonatal hemorrhage, nectrozing enterocolitis of the newborn and chronic respiratory disease originating in the neonatal period to name a few (Callaghan et al. 2006).

Both past and present research has attempted to take into account the methodological complexities involved in modeling the associations between birth outcomes and infant mortality. Birth outcomes are repeatedly recognized as the most important of proximate determinants, strongly influencing various types of infant mortality risks, from timing-of-death to cause-of-death structures (Alexander et al. 1999; Forbes et al. 2000; Powers et al. 2006). The adverse forms of these birth outcomes have been traditionally defined in a dichotomous manner: low birthweight (LBW) [< 2500 grams vs. ≥ 2500 grams] and preterm

(PT) [< 37 weeks gestation vs ≥ 37 weeks gestation]. Recently, efforts have been made to redefine and elaborate the conceptual and methodological linkages between these two crude measures of adverse birth outcomes and infant mortality risk (Alexander et al. 1999; Frisbie, Forbes and Pullum1996; Hummer et al. 1999; Powers et al. 2006; Solis, Pullum and Frisbie 2000; Wilcox 2001; Wilcox and Skjœrven 1992). These efforts spring from the currently expanding knowledge of the underlying complexity of this relationship coupled with the development of new and relevant statistical methodologies in association with advances in computing power.

Virtual complete reliance on categorical measures of birth outcomes has seemingly hampered researchers' goals in explaining race/ethnic differences in infant mortality. Implicit acceptance of a "one-size-fits-all" approach to identifying optimum birth outcome categories based on the experience of Whites has also been a stumbling block toward methodological and substantive progress (Alexander et al. 1999; Solis, Pullum and Frisbie 2000; Wilcox 2001; Wilcox and Skjœrven 1992). An illustrative example is the secular trend in birth outcomes. Paneth (1995) demonstrates that as neonatal mortality fell precipitously from the years 1949 to 1991 in the United States, the low birthweight rate has remained constant, or only slightly decreased, while the preterm birth rate has risen in recent years. Evidence clearly shows that infant survival optima vary widely by sex and race/ethnicity (Gage and Therriault 1998; Kline et al. 1989; Wilcox

2001). Additionally, Alexander et al. point out that "the basic mechanism for the increasing racial gap in infant mortality is racially disparate reductions in both birthweight and gestational age specific neonatal mortality" (1999:77). Numerous authors have commented on the separate roles that birthweight and gestational age may play in influencing infant mortality independent of each other (Alexander et al. 1999; Solis, Pullum and Frisbie 2000; Wilcox 2001; Wilcox and Skjœrven 1992). Many of these same authors have also pointed out that most research in this area has favored birthweight analyses at the expense of gestational age effects on infant mortality risk (cf. Buekens et al. 2000). A few exceptions to this trend continue to show the important and independent adverse effect of preterm birth on infant mortality risk (Kramer et al. 2000), as well as the strong independent and conjoint effects of birthweight and gestational age on infant mortality among White, Black and Mexican American female infants (Powers et al. 2006; Solis, Pullum and Frisbie 2000). Solis, Pullum and Frisbie (2000) and Powers et al. (2006) continue to demonstrate the utility and necessity of analyzing birthweight in both a *standardized* metric form and in combination with gestational age-specific analyses. This string of important findings is strongly linked by one overarching issue: the need to fully utilize continuous measures of both birthweight and gestational age in order to discover the full range of complex relationships that exist within these important causal processes.

This dissertation research seeks to specifically investigate the independent and conjoint effects of birthweight and gestational age across three race/ethnic groups in a more rigorous manner by substituting continuous approaches for categorical ones. The general aims of this dissertation are as follows:

1. Reanalyze the work of Solis, Pullum and Frisbie (2000) in order to assess a more fine-grained and efficient methodology for the analysis of gestational age-specific standardized birthweight distributions by infant mortality.
2. Extend this new infant mortality modeling approach established in Aim 1 to race/ethnic groups other than White females, namely Black and Mexican American female infants, for the years 1989-1991, as a means of comparing race/ethnic infant mortality patterns.
3. Compare the results from Aim 2 to a second time period (1995-1997) in order to ascertain any differences in infant mortality risk.

What follows within this introduction and background chapter is: (1) a review of the historical research and development of birth outcome analyses in order to lay the methodological and conceptual foundations for further discussions; (2) a review of the importance of recent breakthroughs in analytical thinking and how these changes have redefined the conceptualization of this entire research enterprise; (3) a discussion of several complex factors involving

new approaches concerning the utilization of birthweight and gestational age as they pertain to this dissertation's development and conceptual strategy; and (4) a restatement of the general aims as specific aims in order to present a more descriptive and detailed list of the specific analyses that will be undertaken given the background, previous literature and dissertation scope.

1.2 Background and Previous Research

1.2.1 Introduction: "The Story of Birthweight"

The history of infant mortality research has been characterized by the need to accurately measure, model, and control for the powerful effects of birth outcomes on infant mortality (cf. Frisbie, Forbes and Pullum 1996; Solis, Pullum and Frisbie 2000). The results of research on birth outcomes and infant mortality have moved the measurement of birth outcomes from primarily categorical conceptualizations to continuous ones. This history of birth outcomes research within the framework of infant mortality begins with the epidemiologic discovery and understanding of the strong correlation between birthweight and infant mortality in the early 20th century (Kline et al. 1989; Wilcox 2001). From early on, birthweight, specifically *low birthweight*, came to be associated as the most important proxy measure for the concept of *prematurity*, a concept that had been originally defined in 1902 as a birth before 37 weeks of gestation (Kline et al. 1989; Wilcox 2001). Prematurity refers to the basic distinction between infants

that have some aspect of their intrauterine development stunted and/or impaired, thus significantly increasing the probability of infant death within the first year of life as compared to those infants for whom *in utero* development is normal. Due to the difficulty of obtaining accurate gestational age data versus birthweight estimates and the robust findings concerning the adverse infant mortality experiences of low birthweight births, many countries adopted birthweight indicators within their respective vital statistics records (Kline, Stein and Susser 1989). Through the 1950s, at the recommendation of the World Health Organization, epidemiologic studies continued to utilize LBW as a measure of prematurity until the World Health Organization updated their recommendations and urged that LBW not be utilized as the official definition of prematurity. Researchers were quickly beginning to realize that LBW and prematurity were not synonymous and that a reemphasis on the study of gestational age must be highlighted even amidst the difficulties of obtaining accurate gestational age data, both domestically and internationally (cf. Yerulshamy 1967).

Once a consensus among perinatal epidemiologists concerning the importance of utilizing both birthweight and gestational age in identifying high-risk births was achieved, research efforts escalated during the 1960s and beyond. Early work in this area sought simply to identify high neonatal mortality "zones" within the conjoint distribution of birthweight and gestational age (e.g. Battaglia and Lubchenco 1967; Koops, Morgan and Battaglia 1982; Lubchenco, Searls and

Brazie 1972; Yerulshamy 1967). As both theoretical and methodological advances increased, the utilization of multivariate modeling strategies led to a more complex picture of the various associations between birth outcomes and mortality than had previously been presented through mostly descriptive studies.

1.2.2 Extant Issues in Birth Outcomes and Infant Mortality

Of the many substantive issues that derived (and continue to exist) from more complex analyses of birth outcomes (mainly gestational age and birthweight) and their relationship to infant mortality, three important, extant themes/debates serve as special guideposts to this dissertation's specific research aims: 1) the migration from macro-level analyses utilizing aggregate data to analyze rates to micro-level analyses of individual-level data and the subsequent modeling of individual infant mortality risk; 2) the continued emphasis on categorical approaches in lieu of the utilization of continuous data; and 3) the need for conceptual and theoretical clarity in discussing and modeling the complementary link *between* birthweight and gestational age on subsequent infant mortality *risk*, an issue that highlights the extant ideas of birthweight standardization and gestational age-specific analyses, both at the macro- and micro-levels. These three issues form the necessary thrust toward a reformulation of the complex patterns and associations found between birth outcomes. A more thorough discussion of each theme/debate will shed light on the necessity of this dissertation's research direction.

In concordance with the methodological and statistical history of demographic research, the earliest work on the determinants of infant mortality relied heavily on the use of aggregate, or macro-level, data as the primary unit of analysis. Beginning with the analyses of infant mortality rate differentials between nation-states of differing socioeconomic levels (often measured by items such as GNP, unemployment or education rates), these macro-level studies allowed researchers to begin to get a handle on the possible mechanisms and determinants of infant mortality at the individual, or micro-level, by utilizing extant data on population groups, be they differing geopolitical levels or smaller, more "ecological" analyses of census tracts and the like. With increasing knowledge of the strong deleterious effect of extreme poverty and low education on aggregate infant mortality, demographers could begin to postulate possible individual-level scenarios and hypotheses that might someday be testable with the advent of robust micro-level sources of data and both methodological and statistical approaches. This macro-level approach toward infant mortality research continues today and is an integral and vibrant source of knowledge on the ecological distribution of infant mortality vis-à-vis population-level parameters such as the availability and distribution of advanced infant health care technology "down" to the aggregated individual-level characteristics including race/ethnic population composition and maternal education. Of even greater popularity is the resurgence of interest on the specific characteristics of

"neighborhoods" and their "contextual" impact on pregnancy outcomes (Bell et al. 2006; Grady 2006; Morenoff 2003; Subramanian et al. 2006). As can be plainly seen, macro-level approaches to infant mortality research not only set the stage for future work on both levels, but current work focused on the statistically and substantively significant effect of community characteristics on the reproductive health of families has pushed the envelope in the conceptualization of the proximate determinants of infant mortality.

Current research has moved forward in exploring the full, continuous distribution of birthweight in order to identify the components of this distribution and their relationship to infant mortality (Wilcox and Russell 1986; Gage and Therriault 1998; Gage 2000; Wilcox 2001). It has been found that birthweight distributions are primarily Gaussian in nature with a slightly "longer" tail at the low end of the distribution. These authors have focused in great detail on the idea that birthweight is composed of two, separate populations distributions, one Gaussian and one not. The combination of these two distributions produces the current picture of birthweight and its effects on infant mortality. Gaussian mixture models have been applied to aggregate data to model these two distributions and their differential mortality risks (Gage and Therriault 1998; Gage 2000). What is important to understand is the growing acceptance of this dual-distribution analytic picture of the link between birthweight and infant mortality. Given the data integrity issues surrounding the collection of accurate

gestational age data coupled with the ease of access to extremely accurate birthweight data, the detailed study of birthweight with its mix of low and high risk distributions has introduced questions on the fundamental role of birthweight within the causal pathway of infant mortality. Current debates have highlighted this new thinking on birthweight with questions surrounding the popular assumptions concerning its direct effect on infant mortality risk (Wilcox 2001; Melve and Skjaerven 2003; Platt et al. 2004; Hertz-Picotto 2003; Basso, Wilcox and Weinberg 2006). Is birthweight a proper and effective variable for studying infant mortality risk? Is it possible that birthweight, especially at the population level, is not located within the causal pathway of infant mortality? Is it possible that a confounder variable affects both birthweight *and* infant mortality independent of each other? Is it possible that population changes in mean birthweight may not lead to any discernable change in overall infant mortality rates?

Frequent use of aggregate measures of birthweight at the population level have given rise to strong beliefs in the link between birthweight and infant mortality as well as called into question this link. Wilcox (2001) effectively summarizes these issues by displaying and discussing various anomalies within previous LBW studies. Wilcox identifies two examples of where population level use of birthweight as a proxy for infant mortality research have led to incorrect conclusions based on erroneous assumptions. The first anomaly found is in the so-

called “LBW paradox”. This paradox results from the use of aggregate data in order to analyze LBW and infant mortality across different populations. According to Wilcox (2001), historical data have shown a “cross-over” effect in birthweight and infant mortality rates between various subpopulations. This cross-over effect is defined as lower infant mortality rate of infants born at lower birthweights which then crosses as birthweight increases for populations at higher risk of adverse birth outcomes. Three of these populations are high-altitude births versus others, births from mothers who smoked versus non-smoking mothers and Black-White differentials. In each case, Wilcox argues that the fundamental birthweight-infant mortality assumption is erroneous. When Wilcox controls for each population’s specific birthweight distribution, the crossover effect disappears. Wilcox argues that any given variable that defines the subpopulations in relation to each other (e.g. altitude, smoking status, race/ethnicity) may both “push” the entire normally-distributed birthweight distribution to the left as well as increase the overall infant mortality rate. The key is the independence of the relationships discussed. Birthweight is effectively removed as a direct causal agent of infant mortality. The argument is put forth that *maturation* is the key conceptual factor necessary in identifying and understanding high-risk births within the secondary, “residual” birthweight distributions. Usage of the entire birthweight distribution will effectively hide this residual population and lead to an incorrect emphasis on birthweight itself as an important predictor and outcome

variable, one that even if affected positively through public health or medical interventions, may not lead to reductions in infant mortality differentials of importance. What we *must* return to is an increasingly sophisticated approach to studying gestational age as a direct causal link to infant mortality.

The hypothesis concerning the independent roles played by birthweight and gestational age has resulted in much debate within the epidemiological community. Some have responded by accepting Wilcox's statement but questioning the political and social ramifications of this new direction (David 2001). Others have responded against this new concept and questioned both the assumptions and data interpretations (Hertz-Piciotto 2001; Schisterman and Hernandez-Diaz 2006). The latest installment of this debate came last year. Basso, Wilcox and Weinberg (2006) expand their research agenda by simulating infant mortality models in order to examine the effect of confounders on both infant mortality birthweight distributions. Their results show that a confounder can hypothetically influence both infant mortality and birthweight distributions independently. This confounder would have a total prevalence of 0.5% and yet would have profound effects on both fetal growth (a resulting mean birthweight decrease of 1.7 standard deviations) and infant mortality (relative risk=160). They posit a host of possible candidates for this hypothetical confounder: malformations, fetal or placental aneuploidy, infections, or imprinting disorders.

One important key to the aforementioned research is the understanding of the importance of standardizing birthweight distributions in any given modeling scheme. This critical transformation is most forcefully supported within the work of Wilcox and Skjaerven (1992) and Wilcox (2001). What these authors promote is an understanding of the need to control for birthweight distributional differences by transforming the birthweight distribution. They often use z-scores as this method of standardization. From their research by these and other scholars, we find that there are many cases where heterogeneity between birthweight distributions is a result of an overall shift in the mean birthweight for any given subpopulation. The authors show how standardizing for birthweight distributions results in the disappearance in infant mortality rate differentials at the population level. This finding applies to many subpopulation comparison groups, from infants of mothers who smoked (while their infants were *in utero*) versus those who did not to race/ethnic populations and infants born at varying altitudes.

The importance of this approach is key to begin the process of rethinking birth outcomes research. As we move from population-level analyses of birthweight distributions and infant mortality curves to individual-level analyses of comparative infant mortality risk, we must deal with the issue of birthweight standardization. Wilcox and colleagues make strong arguments for a standardization approach that has since been followed usefully by an important precursor work of this dissertation (cf. Solis, Pullum and Frisbie 2000). I continue

in this approach by utilizing standardized birthweight by race/ethnicity in order to "control" for birthweight distributional differences at the population level. What this means is that many researchers, myself included, view this approach as a way of simply controlling for a vast number of variables that have shaped the childbearing experience of mothers from different race/ethnic backgrounds in the U.S. This approach does not mark an explicit or implicit affirmation of the role of genetics in identifying meaningful differences between any race/ethnic groups in the U.S. Race/ethnicity is considered a proxy for various "historical and social processes" that have occurred within the race/ethnic population of interest and have shaped many of its biological and physiological processes.

Percentage distributions, infant mortality rates and ratios of Non-Hispanic Whites, Non-Hispanic Blacks and Mexican Americans: 1990 & 2000

Table 1. Non-Hispanic White Infants

% Births			Pregnancy	IMR* per 1,000 Births		
%Δ	1990	2000	Outcome	1990	2000	%Δ
22.3	0.94	1.15	**VLBW**	308.1	229.5	-25.5
17.4	5.63	6.61	**LBW**	71.2	52.8	-25.8
12.7	1.34	1.51	**EPT (< 32)**	222.0	173.4	-21.9
22.6	8.51	10.43	**PT (< 37)**	46.5	33.0	-29.0
			Neonatal	4.5	3.8	-15.6
			Postneo	2.7	1.9	-29.6
			Infant	7.2	5.7	-20.8

Table 2. Non-Hispanic Black Infants

% Births			Pregnancy	IMR* per 1,000 Births		
%Δ	1990	2000	Outcome	1990	2000	%Δ
6.8	2.95	3.15	**VLBW**	316.1	265.7	-15.9
-1.3	13.34	13.17	**LBW**	87.1	75.6	-13.2
-11.9	4.64	4.09	**EPT (< 32)**	207.1	203.0	-2.0
-7.9	18.90	17.41	**PT (< 37)**	62.2	56.3	-9.5
			Neonatal	11.0	9.2	-16.4
			Postneo	5.9	4.4	-25.4
			Infant	16.9	13.6	-19.5

Table 3. Mexican American Infants

% Births			Pregnancy	IMR* per 1,000 Births		
%Δ	1990	2000	Outcome	1990	2000	%Δ
14.1	0.92	1.05	**VLBW**	308.5	241.4	-21.7
8.9	5.53	6.02	**LBW**	71.3	56.4	-20.9
1.9	1.55	1.58	**EPT (< 32)**	172.6	153.0	-11.4
4.2	10.56	11.00	**PT (< 37)**	35.2	29.0	-17.6
			Neonatal	4.5	3.6	-20.0
			Postneo	2.7	1.8	-33.3
			Infant	7.2	5.4	-25.0

Table 4. Non-Hispanic Black/Non-Hispanic White Infant Ratios

% Births			Pregnancy	IMR* per 1,000 Births		
%Δ	1990	2000	Outcome	1990	2000	%Δ
-12.7	3.1	2.7	**VLBW**	1.0	1.2	12.8
-15.9	2.4	2.0	**LBW**	1.2	1.4	17.0
-21.8	3.5	2.7	**EPT**	0.9	1.2	25.5
-24.8	2.2	1.7	**PT**	1.3	1.7	27.5
			Neonatal	2.4	2.4	-1.0
			Postneo	2.2	2.3	6.0
			Infant	2.3	2.4	1.7

Table 5. Mexican American/Non-Hispanic White Infant Ratios

% Births			Pregnancy	IMR* per 1,000 Births		
%Δ	1990	2000	Outcome	1990	2000	%Δ
-6.7	1.0	0.9	**VLBW**	1.0	1.1	5.1
-7.3	1.0	0.9	**LBW**	1.0	1.1	6.7
-9.5	1.2	1.0	**EPT**	0.8	0.9	13.5
-15.0	1.2	1.1	**PT**	0.8	0.9	16.1
			Neonatal	1.0	0.9	-5.3
			Postneo	1.0	0.9	-5.3
			Infant	1.0	0.9	-5.3

Notes:
VLBW: < 1500 grams birth weight
LBW: < 2500 grams birth weight
EPT (<32): < 32 weeks gestation at birth
PT (<37): < 37 weeks gestation at birth
Neonatal: infant death within first 27 days of life
Postneo: infant death between 28 and 365 days of life
*Infant Mortality Rate (IMR): Number of infant deaths within first year of life divided by number of live births
Source: 1990: 1990 NCHS Linked Birth/Infant Death Vital Statistics Files
2000: National Vital Statistics Report, Vol. 50, No. 12, August 28, 2002

1.3 Current Data

1.3.1 National Statistics

The preceeding tables present a detailed view of the distribution and infant mortality risk of pregnancy outcomes among Non-Hispanic Whites, Non-Hispanic Blacks and Mexican American infants in the United States for the years 1990 and 2000 along with race/ethnic and time-specific distribution and rate ratios. Working from the center leftward in Tables 1 through 3, these columns present the 2000 and 1990 percentage distributions along with the percent change over that time period. On the right side of Tables 1 through 3, mortality rates over time are displayed with the last column representing the percentage change in said rates. Tables 4 and 5 list the distribution and rate ratios between the Black and Mexican American populations versus Whites. The leftmost and rightmost columns represent the % change over time for the aforementioned ratios.

To begin, Whites and Mexican-origin mothers (Tables 1 and 3 respectively) have seen increases in all categorical measures of adverse birth outcomes, with Whites by far experiencing the most negative distributional shifts (left columns) compared to Mexican origin mothers. Between 1990 and 2000, Whites have experienced a 22% increase in the proportion of VLBW births, a 17% increase in LBW births, a 12% in EPT births and an overall 23% increase in PT births. Mexican origin mothers have seen their greatest percentage increase of adverse birth outcomes in the form of VLBW births at 14%. Blacks have

experienced increases in VLBW birth only and have experienced decreases in all other adverse birth outcomes. Although these numbers are for the population of births, the relative rarity of some of these birth outcomes must be taken into account when assessing whether this is a trend in the data or random variability.

Conversely, all groups (Tables 1-3) have witnessed very similar decreases in infant loss, be it in the form of neonatal, postneonatal or total infant mortality (right columns). These decreases range from 15% to 33%. The greatest gain for all groups has been in the reduction of postneonatal mortality.

How is it possible that as the percentage of adverse birth outcomes has increased for two of the three groups listed, while infant mortality statistics showed marked decreases in rates for all groups? The key is the analysis of the adverse birth outcome-specific infant mortality rates in the right columns of Tables 1-3. The increase in adverse birth outcome distributions are more than offset by the decreases in the rate-specific mortality of said outcomes, almost equaling the overall fall in infant mortality.

Tables 4 and 5 bring to light the most important comparisons of the set, especially for Blacks versus Whites. Here, both distribution and rate ratios are calculated using Whites as the denominator versus Black and Mexican American numerators. Some positive and negative trends can be witnessed. On the distribution side, disadvantaged Black distributions vis-à-vis Whites are decreasing in relative difference. In 1990, Blacks disadvantage in the distribution

of adverse birth outcomes relative to Whites (presented as a simple ratio) ranged from 2.2 for PT births to a high of 3.5 for EPT births. In 2000, all B/W ratios had decreased by an average 18.8%, with the highest ratio being 2.7 for both EPT and VLBW. The case for the Mexican American population is, paradoxically, composed of near parity with Whites, with ratios hovering around the 1.0 and 1.2 in 1990 to 0.9 and 1.0 in 2000.

An analysis of rate ratios gives us a more compelling story, as these data tell the tale of the current increasing gap in infant mortality between Blacks and Whites along with important components of that trend. Table 4 presents the B/W case. For all four adverse birth outcomes, Blacks have experienced an increasing rate ratio versus Whites. The greatest rate ratio percentage increases are within EPT (26%) and PT (28%) births, with the birthweight categories following fairly closely behind. Interestingly, it is also in this table that we catch a glimpse of the erosion of the so called "small Black birth advantage", a case where in 1990, Blacks were equal to Whites in their mortality rate within VLBW births (1.0) or had a slight advantage of among EPT births (0.9). By 2000, the slight Black advantage among these most disadvantaged and at-risk births has disappeared, with B/W ratios now solidly above 1.0 at 1.2 for both VLBW and EPT births.

Given the change in the relative standing of both birth outcome distributions and outcome-specific risk, it is imperative that we investigate to the fullest extent possible the statistical relationship between birth outcomes and

infant mortality risk. Given the previous research discussed, it is quite possible that statistical research in this area has been hampered by outdated categorization schemes and methodologies. If this is indeed the case, then some level of damage might be done within the public health policy arena if spurious relationships dominate the discourse concerning infant health and well-being interventions. Use of more refined measures for birth outcomes may well result in a clearer understanding of the sequalae of adverse birth outcomes as well as a more precise understanding of infant mortality and morbidity risk. Epidemiologists and public health demographers have played key roles in the development of more refined measures of birth outcomes in order to avoid mistakes of the past concerning maternal and infant health etiology, as well as, developing a more robust understanding of the relationship *among* birth outcomes themselves. The strict reliance on conventional birthweight and gestational age measures, specifically low birthweight and preterm dichotomies at the individual level, has not only limited our understanding of the inherently complex relationship between two distinct perinatal health characteristics, but has also confused our understanding of more conceptual and substantive domains such as the link between minority status and physical well-being.

1.3.2 Previous Demographic Research

Table 6 shows an example of some of the more commonly utilized demographic approaches to assessing the impact of birth outcomes on individual

infant mortality risk. This table serves as a useful outline for a brief history of the important developments of birth outcome typologies within the last 50 years. Moving from models utilizing only birthweight as the birth outcome of interest in a dichotomous manner to more complex models utilizing both birthweight and gestational age in various combinations, we see improvements in model fit and finer distinctions of differential mortality risk that were previously "hidden" by less refined measures.

The first column details the most utilized birth outcome approach to date. Birthweight is divided into LBW (<2500 grams) and VLBW (< 1500 grams) categories. These cutpoints have been shown to adequately display two important thresholds of infant mortality. When using only LBW, the high risk of infant mortality these infants exhibit versus their "normal" counterparts is clearly evident. An OR of 11.44 indicates on average the deleterious effect of *in utero* underdevelopment on birth outcomes. When VLBW is added, one quickly adduces the nonlinear aspect of birthweight risk as the LBW odds falls to 8.33 and VLBW infants experience 46 times greater odds of infant mortality as compared to infants at birthweights above 2500 grams.

Column 2 reports findings from Frisbie, Forbes, and Pullum's (1996) work on a more rigorous categorical approach utilizing both birthweight and gestational age. They calculate this typology using the Fetal Growth Ratio (FGR) approach developed by Kramer et al. (1989) in order to operationalize a third birth

Table 6. Logistic Regression Analysis of Infant Mortality by Three Birth Outcome Approaches, Anglo Females[a]

	Infant Mortality [Odds Ratios]				
N = 3638333	**LBW/VLBW**		**FFP (1996)**	**SPF (2000)**	
Birth Outcome					
Normal	1.00	1.00	1.00	1.00	1.00
LBW	11.44	8.33			
VLBW		46.41			
Frisbie, Forbes, & Pullum (1996):					
Full Comprom.			20.25		
Light Preterm			11.73		
Light IUGR			9.81		
Heavy Preterm			3.05		
Heavy IUGR			2.47		
Solis, Pullum, & Frisbie (2000):					
Early (E)				1.43	1.30
Late (L)				1.12	1.13
Small (S)				2.31	2.14
Heavy (H)				1.59	1.74
E*S					1.07
E*H					0.96*
L*S					1.00[ns]
L*H					0.98[ns]
Intercept	-5.93	-5.93	-6.13	-7.19	-7.06
–2LL	172525	170146	170335	166872	166537
df	1	2	5	4	8

Source: NCHS, 1989-1991 Linked Birth/Infant Death Files.
U.S. residents, singleton births, ≥ 500g, 28-47 weeks.
[a]Unless noted otherwise, all odds ratios $p \leq 0.01$ | * $p \leq 0.05$ | ns = not significant

outcome, namely infant immaturity, otherwise referred to as intrauterine growth retardation (IUGR). IUGR refers to infants that are underdeveloped relative to their gestational age-specific birthweight distributions, even though they may be defined as normal according to conventional measures of prematurity and low birthweight. These authors develop a more refined categorical typology using the FGR, or the ratio of an observed birthweight at a given gestational age to the mean birthweight of that same gestational age. They identified an infant as IUGR

if the FGR ratio was below 0.85. In sum, we see that certain births that would have been identified as normal in previous schemes ("heavy preemies" and "heavy IUGR") do display higher mortality risks (OR=3.05 and 2.47 respectively) relative to infants identified as normal among these three dimensions.

Column 3 results come from the work of Solis, Pullum and Frisbie (2000) [hereafter SPF (2000)]. The crux of their research involves the use of continuous measures of gestational age and birthweight to estimate mortality risk. The novelty of their approach involves the calculation of a gestational age optimum (in weeks, optimum within 39 and 41 weeks) alongside a standard deviation-based birthweight optimum (1 standard deviation above the mean birthweight for the gestational age optimum) that can therefore be used to identify differential morality risks. Their results (reproduced in Table 6) show the differential mortality risks involved with being born early or late (in weeks) and being born above (heavy) or below (small) the previously mentioned birthweight optimum. Interaction terms also highlight the differential risk associated with younger gestational ages and small weight. For example, infants born 1 week early (i.e. 38 weeks) experience an increased odds ratio infant mortality risk of 30% whereas infants born at their gestational age-specific mean birthweight (i.e. Small parameter equals 1/z-score equals 0) experience a 114% increase in the odds of infant death relative to those at 1+ standard deviation. This model will be explained below.

1.4 New Modeling Approaches

1.4.1 Weights, Standards and Debates

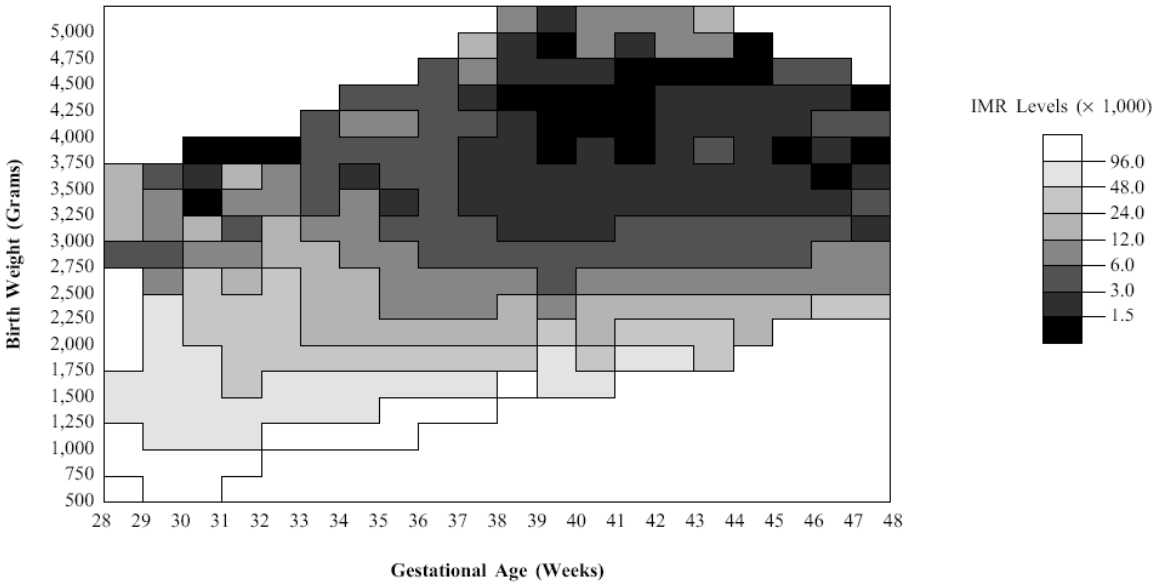
What recent efforts in research of this type have uncovered is the necessity to abandon the idea that all groups' perinatal experiences from conception to birth are equivalent in terms of fetal growth and development. Different historical trajectories coupled with differential socioeconomic and cultural backgrounds heavily influence the perinatal process for different race/ethnic groups in the US. There is little consensus on a standard of measurement that might account for the wide disparity in birth outcomes among various race/ethnic groups. Current thinking now places an emphasis on race/ethnic-specific standards of measuring birth outcomes and modeling their association to infant mortality risk (Frisbie, Forbes, and Pullum 1996; Kline et al. 1989; Wilcox 2001). With a variety of race/ethnic groups exhibiting paradoxical patterns of birth outcomes and/or infant mortality relative to their socioeconomic standing, it behooves current and future researchers to develop and utilize methods that will take full account of these differences and lead to a better understanding of the forces shaping current race/ethnic infant mortality risk differentials (Frisbie, Forbes and Pullum 1996; Frisbie, Forbes and Hummer 1998; Kline et al. 1989; Powers et al. 2006; Solis, Pullum and Frisbie 2000; Wilcox and Russell 1990).

The last model in Table 6 presents a new approach that has been recently used to operationalize birthweight and gestational age and to estimate their

independent and joint effects on mortality risk while retaining a continuous metric (Echevarria 2004; Powers et al. 2006; Solis, Pullum and Frisbie 2000). This novel strategy appears to be an innovation well worth pursuing in the case of sex- and race/ethnic-specific groups other than White females (the only group to which the approach was applied). However, it warrants mention that a major reason for the optimism regarding the potential of this approach to yield new insights is that it facilitates the application of statistical methods that are sufficiently powerful and flexible to rigorously analyze complex issues. Any and all approaches to modeling the birthweight/gestational age relationship depend on locating a point (or points) at which birth outcomes (independently and conjointly) are optimal for infant survival (SPF 2000).

Figure 1. Infant Mortality by Birthweight and Gestational Age: White Females, 1989-1991.

[In: *Demography* 2000;37:489-498]



Source: 1989–1991 NCHS Linked Birth/Infant Death Files.

SPF (2000) began their methodological strategy with a graphic analysis of the patterns of association between birthweight and gestational age (considered conjointly) and infant mortality, using software designed by Vaupel and colleagues (1997) represented by Lexis diagrams. Specifically, they generated a contour plot that depicts the variation in infant mortality rates across birthweight groups and gestational ages. The results of the application of this method are presented in Figure 1. In this graph, infant mortality rates (IMRs) are grouped at different levels for birthweight (250g increments) and gestational age (one-week increments, beginning with the 28th week). The darker areas represent combinations that produce lower infant mortality. Perhaps the most important pattern that emerges from the graph is that the lowest mortality tends to concentrate in a fairly limited combination of birthweights and gestational ages. This combination is bounded by 39 to 41 completed weeks of gestation and birthweights between 3750 and 4500 grams. Infants born within these boundaries register an IMR below 1.5 per thousand. The graph also shows that, with the apparent exception of a combined increase in gestational age and birthweight, any departure from this "optimal region" seems to be associated with rising risks of infant mortality.

This pattern of variation implies that the effect of birth outcomes on infant mortality might be evaluated by assessing how far birthweight and gestational age depart from the combination that produces the lowest infant mortality. However,

there remains the issue of determining how these distances from the optimal combination should be measured. One solution would be to calculate absolute deviation measures in grams and weeks. The drawback to this approach is that it obscures the partial dependency of birthweight on gestational age (Kline et al. 1989; Wilcox and Skjœrven 1992). More specifically, it neglects the likelihood that a similar absolute weight for infants at different gestational ages may reflect a very dissimilar level of risk. This problem can be illustrated by comparing two 2500g infants, one at 30 weeks and the other at 40 weeks of gestation. On an absolute scale, the weight of these two infants is obviously the same, but on a scale relative to the gestational age-specific distribution of birthweight, the former is heavier than the latter. In other words, the premature infant is large for its gestational age, while the full term infant is small for its gestational age. In the SPF (2000) data, the weight of the premature infant is 0.49 Standard Deviations (SD) above the 30 week average, while the weight of the mature infant is 2.21 SD below the 40 week average.

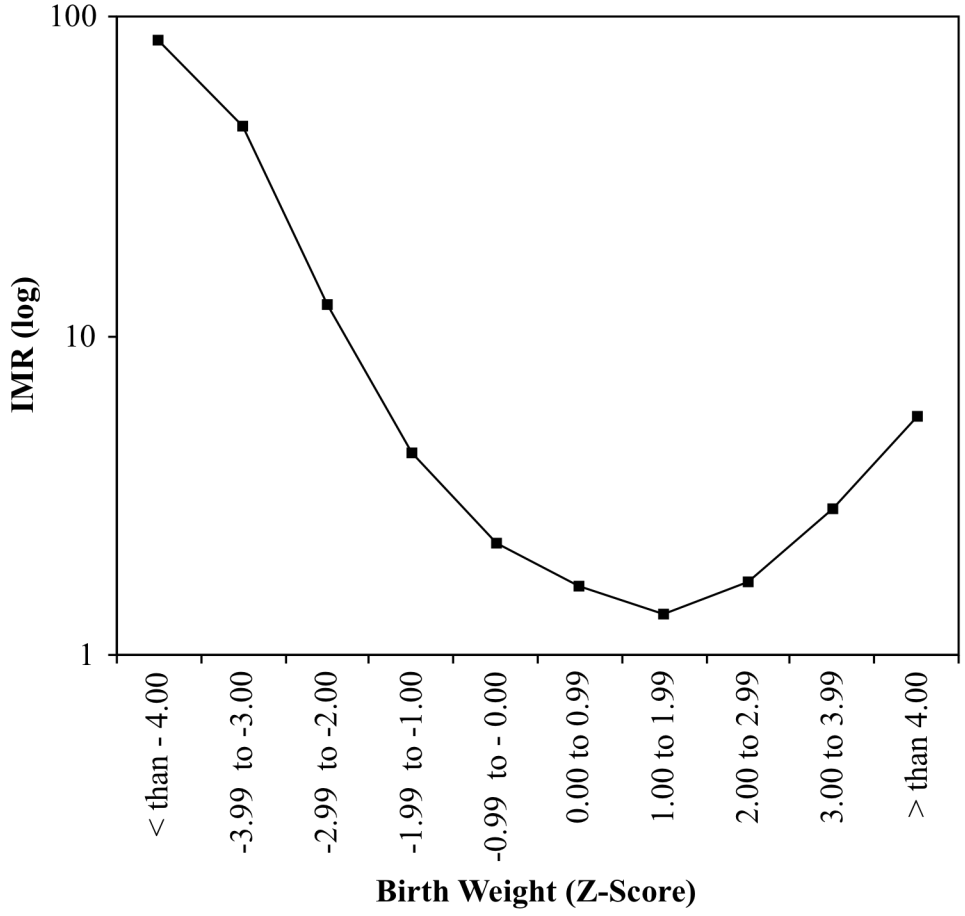
In order to effectively deal with this important issue of relative relationships between birth outcomes, SPF (2000) identify the standardized birthweight point estimate associated with the lowest infant mortality risk among their population of White female infants. This standardized birthweight point was found by transforming infant birthweights into z-scores within the optimal gestational age range of 39-41 weeks. The plotting of z-score birthweights by

infant mortality rates revealed this low IMR point was approximated by 1 standard deviation above the mean for 39-41 weeks gestation (or a z-score of 1). Figure 2 below displays their graph and the rationale for their decision. From the graph one can see the optimum as calculated when aggregating the data into standard deviation groups of one. What is clearly evident is the reverse-J shape of mortality when plotted against birthweight. Also, there is evidence of increasing infant mortality when one moves to heavier weights from the optimum. This optimum lies somewhere between one and two standard deviations. For their purposes, identifying the exact optimum for this group of Non-Hispanic White females born between 1989 and 1991 was not as critical as determining that their was a real optimum and estimating it accordingly for their parameter construction.

The previous steps provide a reference framework for quantifying the distances in birthweight and gestational age from the optimum combination and evaluating their effects on infant mortality. Four separate deviations may be distinguished. First, infant mortality variations may be analyzed as a function of the number of weeks that the delivery precedes the "optimal" minimum of 39 weeks ("Early" or *E*). Second, the effect of gestational age may also be evaluated for post-term newborns by quantifying the number of weeks that the delivery occurs after the 41st week of gestation ("Late" or *L*). The third deviation ("Small" or *S*) reflects the difference on a z-score scale between the optimal birthweight, defined as the mean plus one SD in the gestational age-specific distribution of

Figure 2. Infant Mortality by Birthweight: White Females, 1989-1991

[In: *Demography* 2000;37:489-498]



birthweight, and the weight of infants *lighter* than optimum. Finally, the fourth deviation ("Heavy" or *H*) measures the difference on a z-score scale between the optimal and observed birthweight for infants *heavier* than the optimum. Illustrations of how these distances are obtained appear in SPF (2000). The effect

on infant mortality can be evaluated within the framework of the Generalized Linear Model, by fitting a logistic regression equation of the form:

(1)

$$\log\left(\frac{p}{1-p}\right) = \alpha + \beta_1 E + \beta_2 L + \beta_3 S + \beta_4 H + \beta_5 ES + \beta_6 EH + \beta_7 LS + \beta_8 LH$$

where p represents the infant mortality risk, α symbolizes the logit of the estimated infant mortality risk for infants with the optimal combination of gestational age and birthweight, and the β_i are regression coefficients that summarize the effect of each individual distance and their interactions on infant mortality (Column 3, Table 1). For the full interaction model, the estimated odds of infant mortality (IM) increase 30% (odds ratio=1.30) for every week of prematurity (gestational age < 39 weeks) while the IM odds increase 13% for every week of birth delayed past 41 weeks. The estimated IM odds for a birth whose birthweight is equal to the gestational age-specific mean birthweight (S=1) is 114% higher relative to a birthweight at 1 SD above the gestational age-specific mean birthweight (*S*=0). The shift from 1 SD over the mean to 2 SDs over the mean (*H*=1) produces an increase of 74% in the predicted odds of infant mortality. Important interactions include being both early and small (*E*S*), indicating an additional elevation in risk for premature and relative-low weight infants, and the *E*H* parameter, indicating

a small survival advantage for those premature infants who are heavier than the indicated optimum.

1.4.2 New Standards and Approaches

The novel approach taken by SPF (2000) highlights a new direction in the analysis of individual-level infant mortality risk. As has been stated before, LBW/VLBW approaches are marred by the inability to convey the complexity of the relationship between one measure of infant health, namely birthweight and another, infant mortality. Given the long historical background of this approach, it is appropriate to understand that these simple categorizations may form the backbone of future research in this area, as they are the first attempts at modeling categorically what is inherently a continuous process. The use of VLBW was a watershed development in that it affirmed to all health scientists what perinatologists had long known, (i.e. the non-linear relationship between weight and risk at the individual level). Moving from the simple to the more complex, Frisbie, Forbes and Pullum (1996) made strong strides in illuminating the complexity and nuances found within birth outcomes and their relationship to mortality risk. Although previous researchers had identified, both in theory and practice, the need to also identify those infants that had grown too large for their relative position in the intrauterine growth environment (cf. Kramer 1989), FFP build upon other's work to create a complex categorization schema that displays great diversity in identifying infant mortality risk. SPF continue this line of

thinking by extending the idea of "optimal targets" of birth outcomes and measuring deviations from this optimum as a way of gauging with greater precision the independent effects of both birthweight and gestational age. SPF's work is one of the first important steps at recognizing the essential need to model infant mortality at the individual level *as well as* disentangling the independent effects of birthweight and gestational age. The SPF model allows for careful analysis of individual birth outcomes on both a continuous and categorical scale. This is key in that it allows for simple logistic regression approaches to result in much more complex models of infant mortality odds where independent, main effect terms are coupled with logical interaction estimates in order to create a more powerful predictive model.

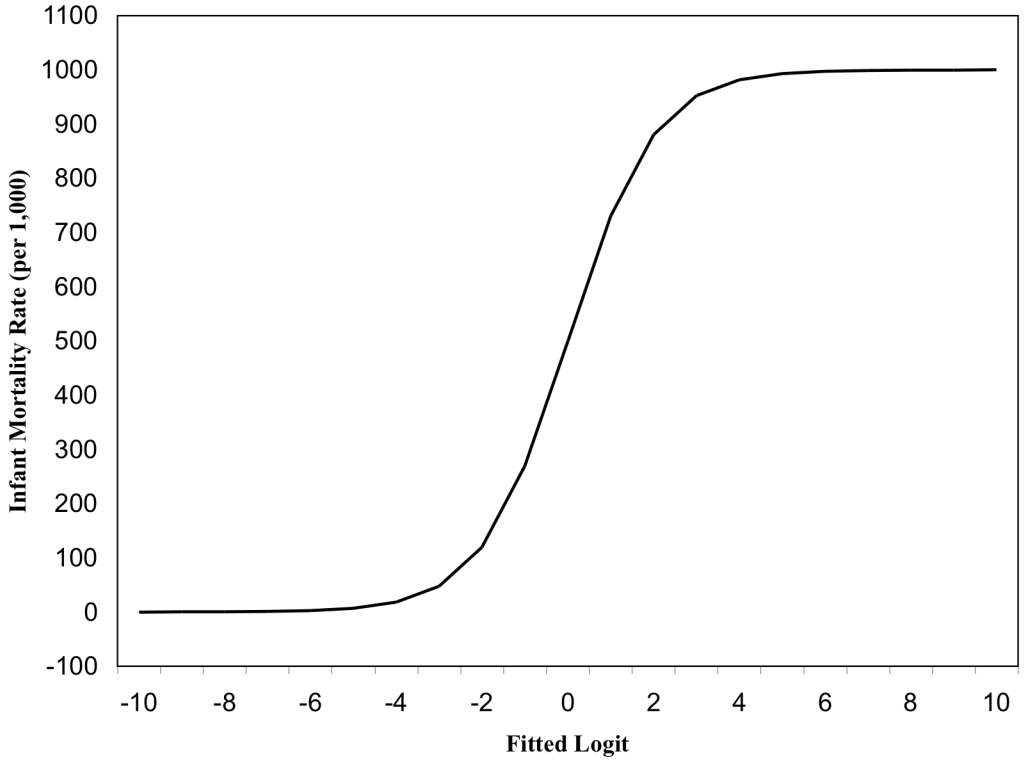
What is clearly evident from the insightful research conducted by these and other authors is the explicit desire to somehow deal with the nonlinear relationship found between both birthweight and gestational age and subsequent infant mortality. By utilizing the full, continuous data after identifying an optimum birthweight, SPF and others allow for interaction and nonlinearity within these distributions to be modeled and displayed. A key issue specified by SPF is how the "design of more adequate methods for identifying the optimal birthweight and exploring its location at different gestational ages is an important topic for further research." (SPF 2000:497). The identification of the race/ethnic-specific optimum is critical in more efficiently modeling birth outcomes

parameters. Given the reverse-J shape of birthweight/infant mortality curves within almost all gestational ages, there is strong evidence that an optimum-based approach has the necessary methodological assumptions to better estimate birth outcome effects on infant mortality.

One component of this study is the mathematical estimation of proper optima for any given subpopulation of interest (e.g. race/ethnicity, sex and time). As SPF state, "Defining the location of the exact optimal relative birthweight is outside the scope of this paper, but we assume for modeling purposes that this optimal point is located one *SD* over the mean (*z*-score = 1)." (SPF 2000:492). SPF identify this optimum for White females after visually inspecting aggregate data on birthweight and infant mortality rates for data grouped into SD categories of one among gestational ages 39 to 41 weeks. What is needed is an alternate, empirical, strategy for identifying optima based on individual-level data in order to identify nonlinear individual *risks* of infant mortality by birthweight. Such a strategy may be found in nonparametric regression models (Hastie and Tibshirani 1990). These nonparametric models do not make any *apriori* assumptions on the mathematical relationship between two variables being modeled against each other. More traditional statistical modeling approaches do make these assumptions based on the level of measurement and other characteristics of the variables. In this case, the most utilized approach for modeling infant mortality, a binary variable, is logistic regression. Logistic regression assumes a parametric

form relationship between variables being modeled, namely that of an

Figure 3. Infant Mortality Rate by Fitted Logit



exponential function. Figure 3 displays this familiar parametric function using the predicted logits and infant mortality rates. What this figure shows is the constraint any logistic regression model has in modeling the relationship between a continuous independent variable and a dichotomous dependent variable. The issue is whether this form is the best representation of the data. Of course, there are important reasons for the use of logistic regression in modeling a binary outcome over other approaches, but for our purposes, it is necessary to test the assumptions

of parametric relationships between increasing birthweight and decreasing infant mortality within a given gestational age, for that would be the constraint and outcome of using logistic regression to find the optimum statistically when using individual-level data. Nonparametric regression methodologies do not utilize any assumed parametric relationship between variables of interest. One nonparametric approach of interest is what is termed the "Generalized Additive Model" or GAM. These more flexible regression techniques allow researchers to explore the actual relationship between exposure and effect. As authors Hastie and Tibshirani state in page one of their textbook on GAMs, "Let the data show us the appropriate functional form" (Hastie and Tibshirani 1990:1). GAMs involve generalized multiple regression techniques (via scatterplot smoothers, and backfitting and local scoring algorithms) to estimate nonparametric functions of independent variables and their additive contribution to the conditional responses/expectations of a dependent variable, in this case the logit of the response probability, generally expressed as:

$$(2) \qquad \log(p_i/(1-p_i)) = \alpha + f_1(x_1) + f_2(x_2) + \cdots + f_j(x_j)$$

or specifically:

$$(3) \qquad \log(p_i/(1-p_i)) = \alpha + f_1(\textit{z-score of birthweight}) \mid \textit{gestational age}$$

where p_i (i=1,…,n) denotes the infant mortality risk as described in SPF (2000).

Backfitting and local scoring, or iteratively reweighted least squares (IRLS), are used to find the net contribution of each of the $f_i(x_i)$'s (fitted by any number of scatterplot smoothers) to the predicted logit. The end result is a set of functions that can be plotted to gain insight into the form of the relationship between birthweight Z scores and infant mortality risk for each gestational age. For my purposes, this stage will involve the modeling of birthweight by gestational age z-scores and their associated infant mortality risk utilizing both normal logistic regression and GAMs. Predicted probabilities are analyzed for each gestational age and race/ethnic group of female infants in order to determine the combinations of standardized birthweight and gestational age that exhibit the lowest infant mortality risk.

Model goodness-of-fit statistics will be compared to find the most efficient and informative model (Figueiras and Cadarso-Suarez 2001). Multiple model chi-square and log-likelihood tests can be performed that facilitate correct specification of the models via nonparametric assumptions versus all other possible permutations of linear/nonlinear terms (e.g. the case where $f_j(BWT_i)$ reduces to $\beta_j(BWT)_i$). One of the most common procedures for estimating model fit via approximations of prediction error is Akaike's Information Criterion or AIC (Hastie and Tibshirani 1990). AIC values can be calculated for both

parametric and nonparametric models and, along with likelihood ratio tests, can used to select the optimal type of model and the appropriate degrees of freedom for nonparametric models (Figueiras and Cadarso-Suárez 2001).

The lack of a single point estimate and standard error can be discomforting; the slope (or effect) of a covariate could be calculated at any point on the fitted function. Because of this, it is necessary to examine plots of the fitted functions and their 95% confidence interval bands against the predicted logit. If a plot is approximately linear (i.e. a straight line could be fitted through the CI bands whereby the software indicates the nonparametric assumption is not statistically significant), the slope would be constant, and the usual linear term for that variable could be included in either future GAMs or standard logistic regression. If the plot is U-shaped, it would indicate the presence of linear and quadratic terms; if the plot is reverse J-shaped, it would imply a log-transformation, and so on. In any case, the GAM model may provide insight into possible *parametric* functions of the covariates that could be used to provide a better fitting model in the context of the proposed methodology. The effects of these deviations on infant mortality can then be evaluated using logistic regression models to estimate the main and conjoint effects on infant mortality for each race/ethnic-specific population.

Given the aims discussed in section 1.1, I can now list how these specific aims will be accomplished within this dissertation:

1. Utilize GAMs to calculate the gestational age-specific optima for all gestational ages and for the gestational age grouping 39 to 41 weeks among U.S. Non-Hispanic White female infants born between 1989 and 1991.
2. Construct the Solis, Pullum and Frisbie (2000) birth outcome parameters for the aforementioned population group using the new optima as the cutpoint for the birthweight-dependent S and H parameters.
3. Reproduce the methods and outcomes in Aims #1 and #2 for two other population groups, namely Non-Hispanic Black and Mexican American females infants born between 1989 and 1991.
4. Reproduce the full logistic regression SPF model including all groups with race/ethnicity as a covariate and unique GAM-calculated optima.
5. Run race/ethnic-specific models for each group in order to test the birth outcome estimates across groups.
6. Lastly, reproduce all previous work for infants born between 1995 and 1997 in order to compare over-time trends and differentials, if any.

CHAPTER 2: DATA AND METHODS

2.1 Data

2.1.1 Data Files

The data for the analysis will be drawn from the 1989-91 and 1995-97 NCHS Linked Birth/Infant Death Cohort Files (U.S. DHHS 1995, 1996, 1999). The data sequence begins in 1989 because it was in that year that a new standard certificate format came into wide use that differs from its predecessors in important ways: (1) It provides specific Hispanic identifiers for all states of the U.S. (except New Hampshire) and the District of Columbia, instead of just the 23 states (and the District of Columbia) reporting Hispanic ethnicity prior to 1989. (2) It includes formerly unavailable items on maternal weight gain, smoking, consumption of alcohol, maternal medical conditions, previous pregnancy loss, and broadened the coverage of education to all states, except New York. (For that state, data are available for New York City.) (3) An item giving a clinical estimate of gestational age was added which allows comparison with (and substitution for) the sometimes suspect estimates of gestational age calculated as the difference between date of birth and date of last normal menses.

Issues surrounding data analyses of this type utilizing national vital statistics records mainly involve the exclusion of certain records. Cases have

Table 7. Births and Infant Deaths by Race/Ethnicity: NCHS Linked Birth/Death Files

TIME	**1989-1991**			**1995-1997**		
Birth Outcome	Births	Deaths	IMR*	Births	Deaths	IMR*
Female Infants						
NH-White	3,771,113	18,088	4.8	3,380,854	13,164	3.9
NH-Black	945,330	10,067	10.7	830,406	6,579	7.9
Mexican American	527,463	2,635	5.0	684,233	2,682	3.9
Total	5,243,906	30,790	5.9	4,895,493	22,425	4.6

Source: NCHS Linked Birth/Infant Death Files. U.S. residents, singleton births, BWT ≥ 500g, GA 22-47 wks.
*per 1,000 live births

been traditionally excluded for two main reasons: (1) in order to reduce data coding errors and (2) to specify a specific population that has been *a priori*

defined as the study population necessary to efficiently model relationships of interest. The first rationale would involve limiting cases within certain pre-specified parameters. When researchers want to study live births, sometimes records with birthweights less than 500 grams are deleted in order to excise errors in recording birthweight (cf. Frisbie, Forbes and Pullum 1996). Records under 22 weeks of gestation may also be deleted for the same reasons. The second rationale involves limiting the population by certain parameters in order to model a "clean" relationship utilizing the most parsimonious approach possible.

When studying the effects of birth outcomes on infant mortality, some researchers choose to limit populations to singleton births by excluding all plural

birth records (cf. Frisbie, Forbes and Pullum 1996). Plural birth infants are known to be more at risk of adverse birth outcomes. Many researchers focus on primiparas in order to isolate a more stable population without the need to identify plural births within modeling.

Lastly, race/ethnicity is measured by conventional methods. This involves NCHS recommended criteria utilizing mother's race/ethnicity as a proxy for infant race/ethnic identification (cf Rogers 1989).

Table 7 shows the frequency of infant birth and deaths along with corresponding infant mortality rates for the three populations of interest and time period. A total of over five million births were used within the period one analysis and just under five million for the second period analysis. From the IMR column, we see both the two-to-one Black-to-White infant mortality disadvantage as well as the Mexican American and White parity in IMRs. At first glance, some important race/ethnic differences appear within this table. First, Whites dominate the birth and death totals through both time periods. Although Whites account for roughly four times as many births as Blacks, they only account for twice as many deaths. This proportional difference is presented in the infant mortality rates by race/ethnicity and time period. Whites exhibit a 2-to-1 survivorship advantage over Blacks that narrows slightly from period one to period two. As has been stated before, Mexican American infant mortality is roughly equal to that of Whites, a paradox easily seen within this data. What is also of interest is the

narrowing of the aggregate number of births between Blacks and Mexican American women. From period one to period two, the percentage of Mexican American births rose as a ratio to Black births from 56% to 82%.

Table 8 displays the race/ethnic distribution parameters of interest for this study for time period one. Mean birthweight is highest for Whites and Mexican Americans, while Blacks exhibit much lower mean birthweights and a higher standard deviation. This displays the distributional differences between race/ethnic groups that is the impetus for much debate in this research area. This Black disadvantage is also seen within the measures for gestational age. Blacks exhibit a one-week differential between mean gestational age at birth compared to Whites and Mexican Americans. These simple measures of central tendency and dispersion underlie fundamental differences in the pregnancy process and sociohistorical trajectories of these groups within the U.S.

2.1.2 Trends

Table 8. Birth Weight and Gestational Age Distributions by Race/Ethnicity – Females

Race/Ethnicity	Mean BWT	SD BWT	Mean GA	SD GA	N	%
NH-White	3367.6	534.3	39.5	2.3	3,771,113	65.2
NH-Black	3077.1	598.0	38.5	3.2	945,330	16.3
Mexican American	3319.7	516.5	39.3	2.5	527,463	9.1

Source: NCHS, 1989-1991 Linked Birth/Infant Death Files.
U.S. residents, singleton births, BW ≥ 500g, GA 22-47 wks.

Figure 4. Proportion Births and Death by Gestational Age:
U.S. Residents, 1989-1991

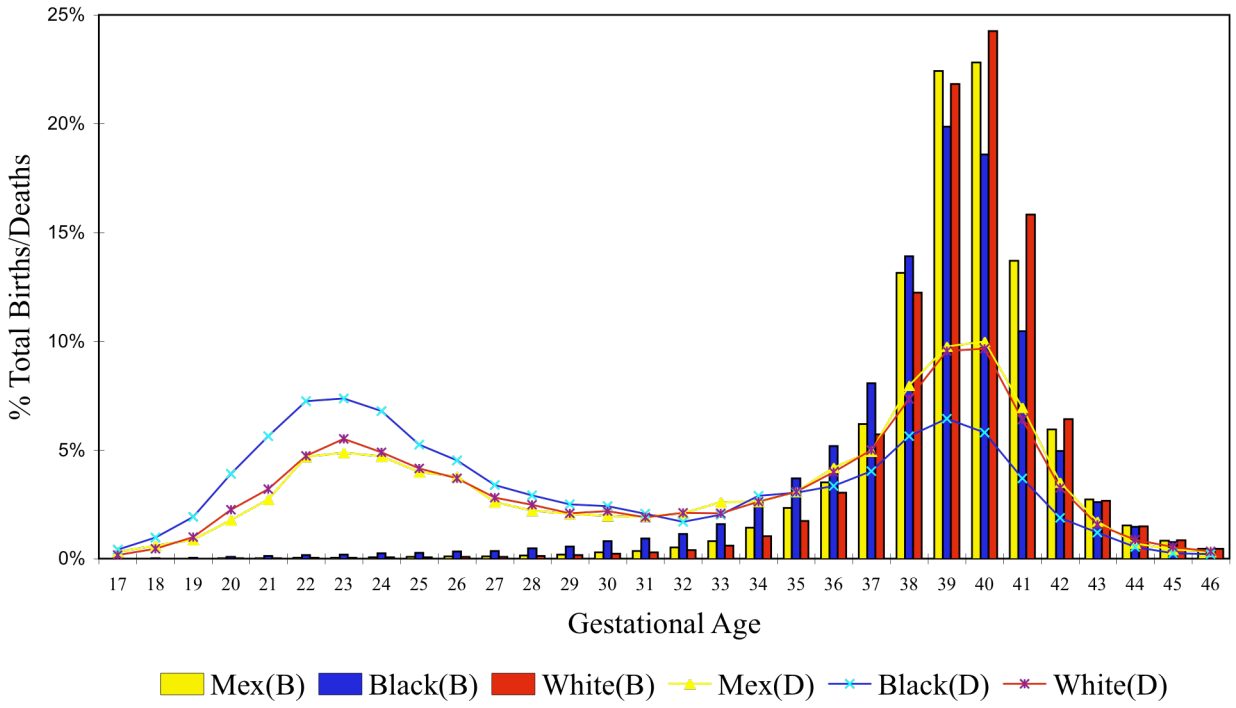


Figure 4 shows the distribution of both births and deaths by gestational age for time period one among all female race/ethnic groups studied. What is striking is the well-known fact that, although the overwhelming number of births occur within a small range of gestational ages (36 to 42 weeks gestation), there is a bimodal distribution of infant death among both early and late gestational ages. What is also clearly visible here is the Black disadvantage in terms of proportion of birth born outside the "normal" range for the human population as well as the higher proportion of deaths at the lower gestational age "hump" in the bimodal

distribution. This graph serves to solidify the natural human birth outcome experience in terms of where births and deaths are concentrated given a 40-week gestation period. It is imperative to remember that infant mortality is composed of both endogenous and exogenous causes of death, so this graph is actually pointing to the effect of both adverse birth outcomes and social context in determining infant mortality risk. As infant mortality is a useful proxy of infant disadvantage at birth and has been extensively utilized for this reason, I will continue this tradition for methodological reasons with this caveat in mind. What also stands out in Figure 1 is the bimodal distribution of infant deaths within very low gestational ages (22-24 weeks) and those centered around 40 weeks gestation. For Whites and Mexican Americans, the largest proportion of deaths occur at 40 weeks while for Blacks, the largest proportion of deaths occurs at 22 weeks. As previously mentioned, Blacks at this level of analysis do exhibit a mortality advantage at low gestational ages. This is less clear within this graph, but is still evident if looked at closely. The key is the distribution of births at these low ages for Blacks (middle bar). From 38 weeks and before, Blacks experience the largest proportion of births at every week as compared to the other two race/ethnic groups. From 39 weeks onward, Whites and Mexican Americans display stark advantages in proportions of births found at these "normal" ages. Also of note are the near identical distributions of Whites and Mexican Americans, a paradox indeed given the disadvantaged SES profile of Mexican Americans relative to

Whites. Births are fairly normally distributed with a mode of 40 weeks for Whites and Mexican Americans and 39 weeks for Black infants.

Figure 5 displays mean birthweights and their associated standard deviations by gestational ages for my study populations. There exists an almost linear increase in mean birthweights as gestational age increases with some leveling off after 40 weeks. Standard deviations increase as well but plateau at 28 weeks, possibly indicating differing sequalae for preterm birth before and after 28 weeks gestation. Although somewhat difficult to see, the blue mean birthweight line of Blacks is slightly higher from the lowest gestational age recorded until around 34 weeks, where the Black "crossover" occurs. From this point onwards, Black lose their slight advantage in mean birthweight and have consistently lower mean birthweights. This disparity in mean birthweight between Blacks and the other two study populations, especially Whites, tends to widen as gestational age increases. If indeed, extant debates notwithstanding, birthweight is directly linked causally to endogenous infant mortality, one can easily see the problem of using arbitrary birthweight optimum cutpoints as thresholds for identifying high-risk birth outcome categories.

Figure 5. Mean Birthweight and Standard Deviations: Females, U.S. 1989-1991

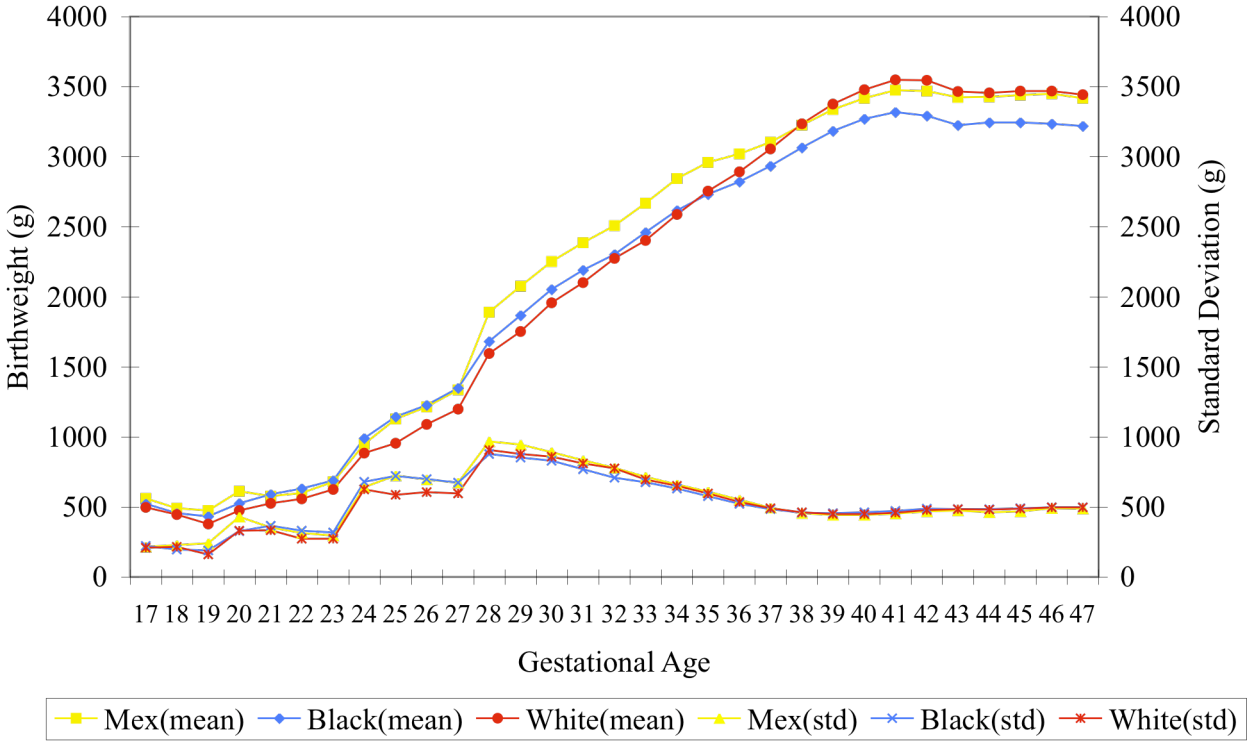


Figure 6. Infant Mortality Ratios by Race/Ethnicity and Gestational Age: Females, U.S. 1989-1991

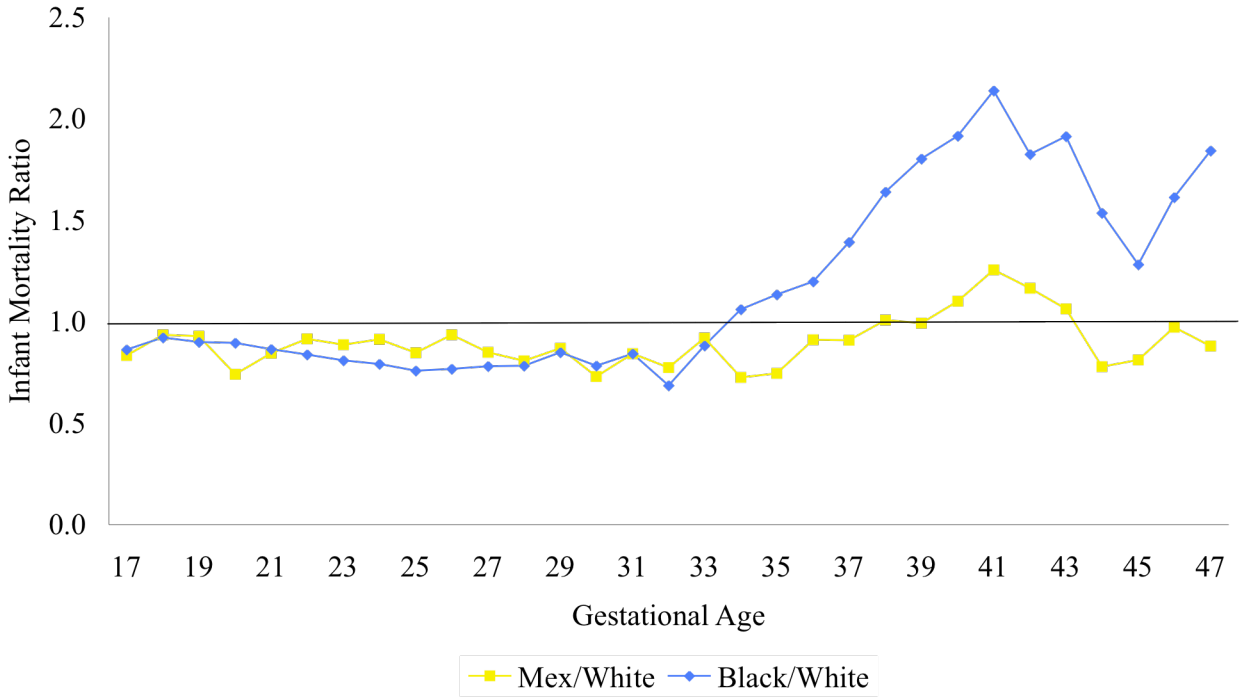
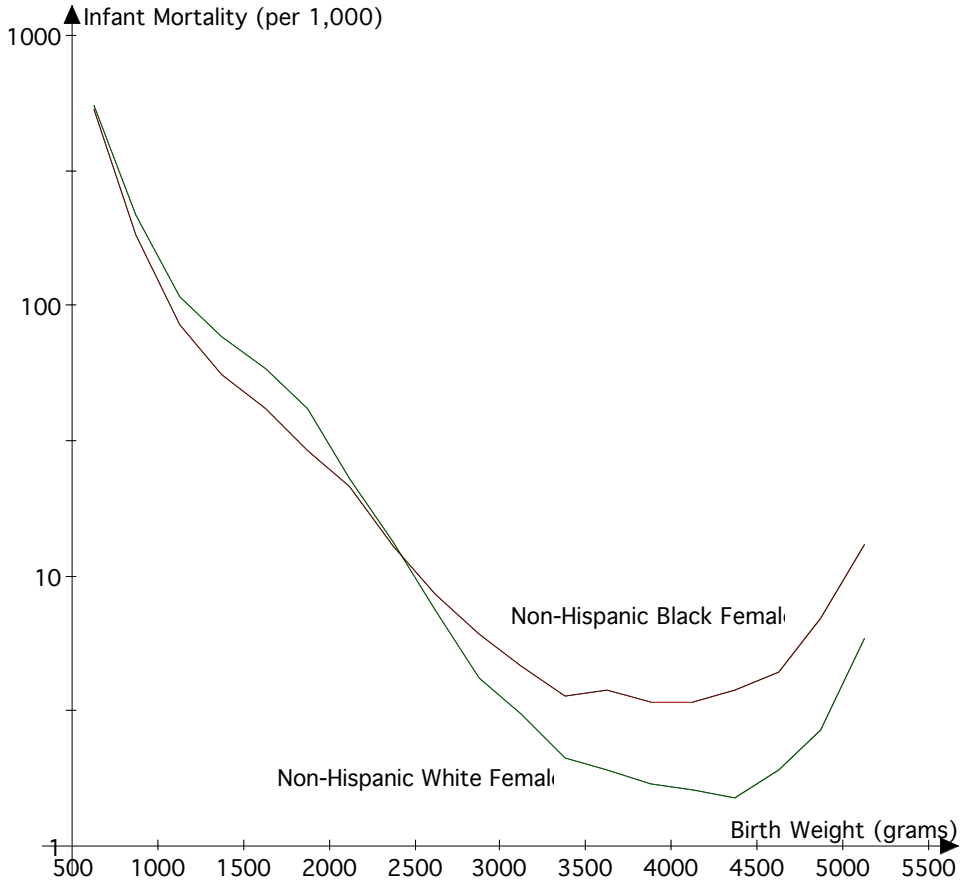


Figure 6 displays the infant mortality data by gestational age in terms of rate ratios relative to Whites. Here, two trends are powerfully displayed, namely the "Epidemiologic Paradox" of Mexican Americans and the Black/White "crossover". The rate ratio for Mexican Americans is almost always less than one, with a slight increase above one for weeks 40 through 43. For Blacks, the rate ratio is consistently below one from 17 to 33 weeks gestation. At 34 weeks and beyond, the rate ratio quickly increases, reaching a peak at 41 weeks of 2.1 and oscillating between 1.3 and 1.9 from 42 weeks onward.

Figure 7. Infant Mortality by Race/Ethnicity and Birth Weight: 1989-1991

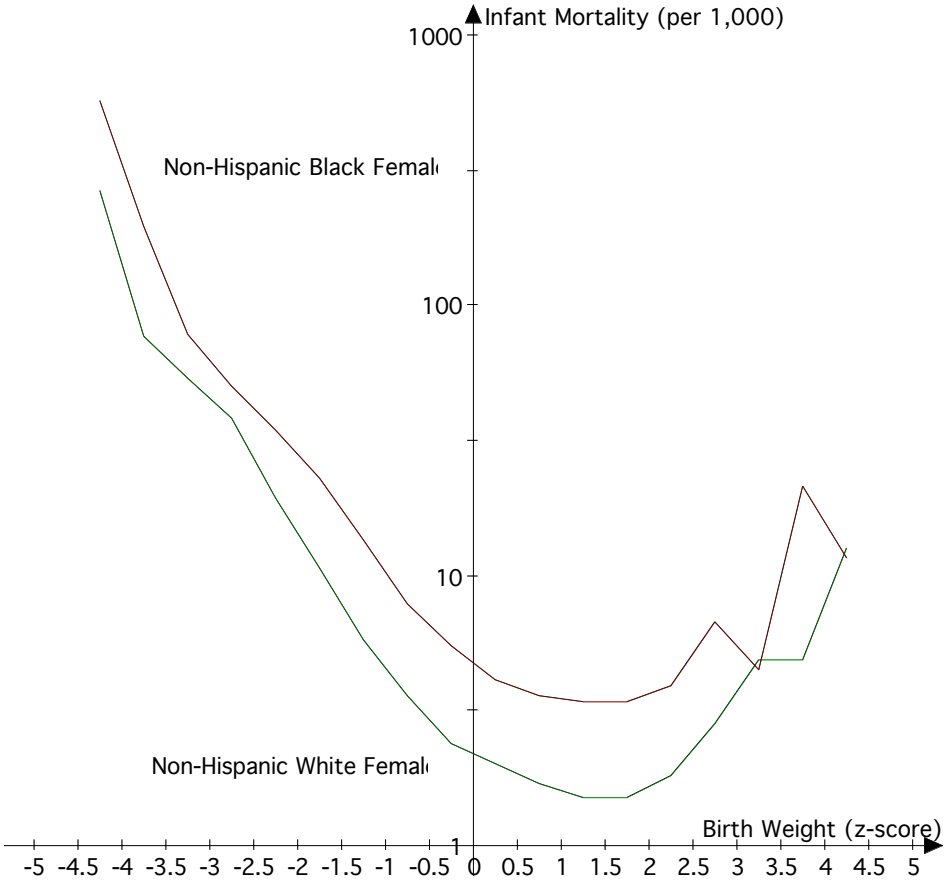


2.1.3 Birthweight, Gestational Age and Infant Mortality

In order to assess the importance of standardizing either birthweight or gestational age in an attempt to gain a clearer understanding of the relationship between the two, Figures 7 through 10 were created. These graphs display the relationship between birthweight, gestational age and infant mortality by race/ethnicity (non-Hispanic Whites versus non-Hispanic Blacks) for the U.S. in

the years 1989-1991. In each set of graphs, we see how standardizing birth

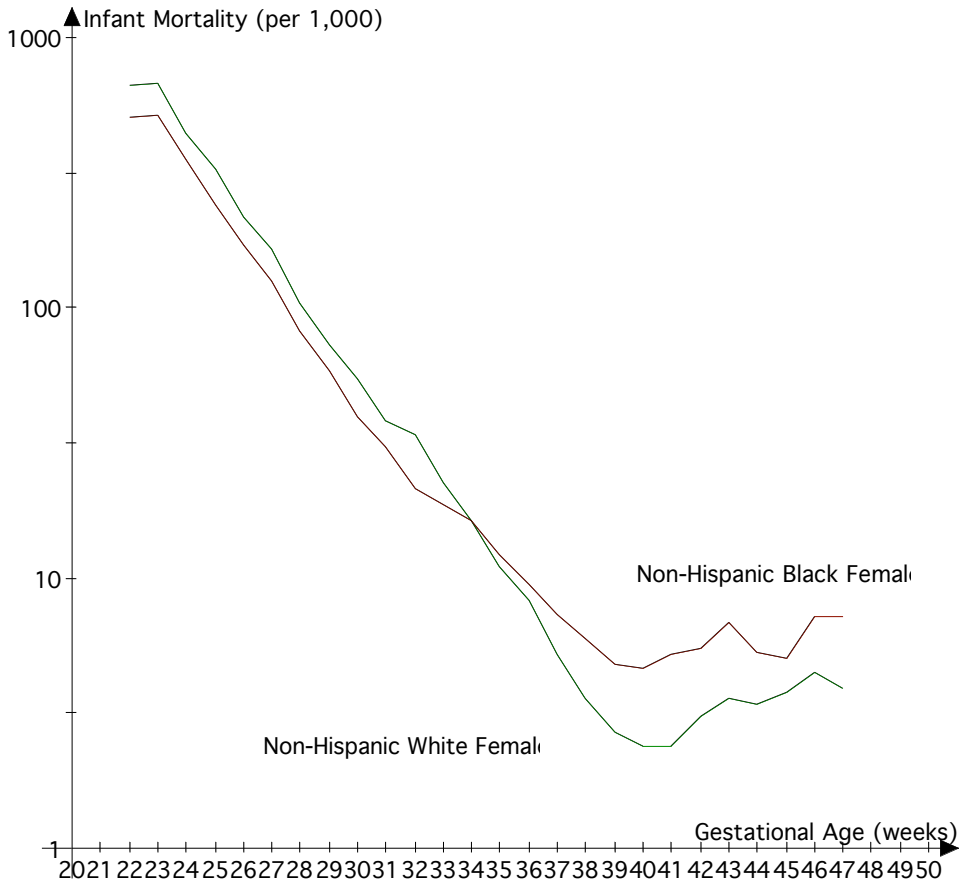
Figure 8. Infant Mortality by Race/Ethnicity and Birth Weight: 1989-1991



outcomes eliminate any and all non-Hispanic Black "advantages" in infant mortality as compared to non-Hispanic Whites. Figure 7 displays data for females. Just as many have shown before, non-Hispanic Black infants possess a mortality advantage between 2,000 and 2,500 grams as compared to non-Hispanic Whites infants. What is also readily apparent is the large disadvantage that Blacks display at heavier weights, from 2,500 grams onward. Figure 8 graphs the

same data transformed into z-scores using each race/ethnic groups mean birthweight as z=0.

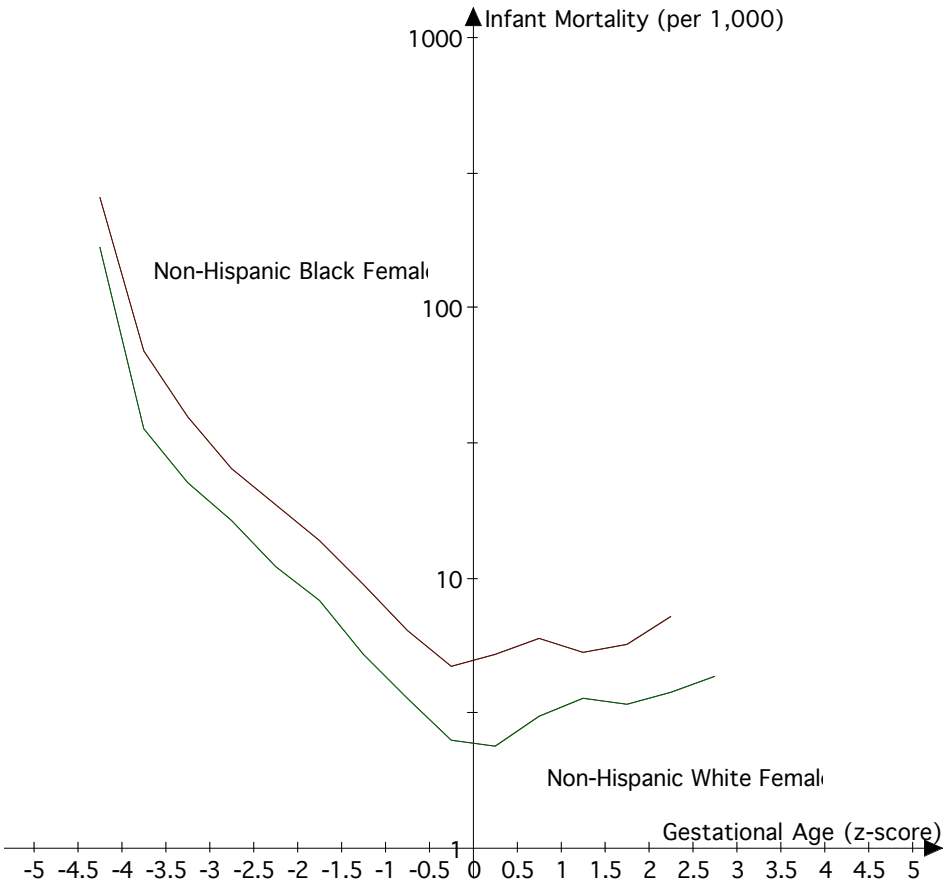
Figure 9. Infant Mortality by Race/Ethnicity and Gestational Age: 1989-1991



When this transformation is utilized, at every level of standardized birthweight, Blacks possess a distinct disadvantage that grows from the lowest z-scores to the highest. At the positive extremes, small cells contribute to the irregularity of the graph. Figures 9 and 10 repeat the same procedure utilizing gestational age data in lieu of birthweight data. Again, the same pattern emerges

where unstandardized data results in a Black advantage at earlier gestational ages but standardized data show a Black disadvantage at all gestational ages.

Figure 10. : Infant Mortality by Race/Ethnicity and Gestational Age: 1989-1991



2.2 Optimum Models

Above, I have illustrated an important approach that allows specification of main and interaction effects of gestational age and birthweight on infant mortality evaluated in terms of their deviation from a combination of gestational

age and birthweight that results in the lowest levels of infant mortality risk. Following the approach applied in their study of the relationship between birth outcomes and infant mortality among White females (SPF 2000), I begin by utilizing both logistic regression and the Generalized Additive Model (GAM) to statistically identify the gestational age-specific birthweight levels that exhibit the lowest infant mortality risk. I quantify birthweight relative to gestational age in terms of deviations from a baseline (z-scores) for all gestational ages. Whereas SPF (2000) utilize the same standardized point estimate (z-score = 1) as a relative birthweight optimum for all gestational ages (and implicitly all groups under study if there were more), I calculate a different relative optimum for each race/ethnic group in order to assess the differential nonlinear/nonparametric mortality risks that may exist at certain gestational ages.

The choice of 1 SD above or below the mean was arrived at iteratively and provided a relatively good fit to the data for White females. There is plenty of evidence to support the hypothesis that this standard is not only not the best fitting optimum for every gestational age among White females, but also is not the best fit for many other race/ethnic groups, even at optimum gestational ages (39-41 weeks) (Alexander et al. 1999; Frisbie, Forbes, & Pullum 1996; Solis, Pullum, & Frisbie 2000; Wilcox and Russell 1990; Wilcox and Skjœrven 1992). Furthermore, there may well be inflections in the curve, other than the optimum point, that denote important survivability changes, including the increase in the

risk of mortality for heavy weight infants. GAM estimation offers an objective means of avoiding a cumbersome iterative procedure—one that might well have to be repeated if survival curves vary in more than a modest way between 1989-91 and 1995-97.

The general methodology of my subsequent modeling approach is discussed in detail below. Utilizing the z-scored birthweight distribution for each group (race/ethnicity and time) within the gestational age range of 39-41 weeks, I ran GAM models in order to obtain the GAM fitted (predicted) logits and their corresponding birthweight (z-score). The smoothing method utilized was a spline smoother with the value of the smoothing parameter chosen by generalized cross validation (method=gcv). Cross validation works by leaving points (x_i, y_i) out one at a time, estimating the squared residual for smooth function at xi based on the remaining n-1 data points, and choosing the smoother to minimize the sum of those squared residuals. Examples of degrees of freedom for both time periods range from a low value of 4.7 for Blacks in period one to a high of 10.1 for Mexican Americans in period two. All models result in significant nonparametric trends.

(I only included z-scores ranging from -4 to +4 sd for all groups. SAS was not able to handle the full distribution for non-Hispanic Whites, so I had to limit the distribution for all groups accordingly in order to minimize any bias at the extremes. Since my intent was to discover optima, this was an acceptable data

limitation. For example, -4 to +4 SD roughly equals from 2-3lb to 12-13lb births for non-Hispanic Whites while the mean lies somewhere between 7-8lbs.)

I then entered the z-scores and their predicted logits into a graphing program that also calculated non-linear algebraic equations for any data entered (Advanced Grapher [AG]) and plotted their relationship (Figure 6). Using AG, I was able to fit an 8^{th}-order polynomial function to the graphed data for each group. Figure 6 displays the results of this process for the groups studied. What is evident is the nonlinear shape of each curve. The models for each group produced by SAS PROC GAM are significant at the $p<0.01$ level, indicating a good fit of this population data to the parameters of the GAM. Within this gestation age range, predicted infant mortality risk falls as one moves from the lightest infants born at -4 SD from their race/ethnic-specific, gestational age-specific (39-41 weeks) mean. For Whites, there is a gradual decrease in risk that does reach an optimum point and then gradually increases from there onward. For Blacks, there seems to be a leveling off of risk once the optimum is reached while Mexican Americans seem to exhibit a true optimum with a sharp increase in risk as heavier infants are born. It is difficult to identify these optima visually, hence the use of AG to plot and produce the fitted, multivariate, nth-order equation for optimum verification.

An example equation is listed below for Figure 6: Non-Hispanic White females, 1989-1991:

$$y=(6.87\bullet10^{-6})x^9+(3.58\bullet10^{-5})x^8-(3.01\bullet10^{-4})x^7-(1.42\bullet10^{-3})x^6+$$
$$(4.89\bullet10^{-3})x^5+(1.65\bullet10^{-2})x^4-(3.22\bullet10^{-2})x^3+0.10x^2-0.37x-6.29$$

The R^2 for this equation equals 0.9999806. Using these equations, I then calculated the mathematical XY minimum pair where the slope equals zero, or where the infant mortality risk is at its lowest. In this case, it is (1.514, -6.618) for Whites. Hence, the optimum for Whites is 1.51 SD above the mean (with a resulting IMR of 1.3/1,000 births). From here, I rescored the full birthweight distributions to z-scores with the new optima at z=0 and created the SPF birthweight parameters. Table 9 below displays the new optima derived from this protocol (the results for this table will be discussed in more detail in the next chapter). For the gestational age parameters, instead of deviations in weeks, I transformed this distribution into z-scores with 40 weeks at z=0. This method was undertaken in order to parallel the S/H parameters as deviations based upon the standard z-score.

Table 9. Optimal Birthweight (z-score) and Infant Mortality

TIME	1989-1991	1995-1997
Gestational Age	39-41 weeks	39-41 weeks
Females		
NH-White	1.51	1.42
NH-Black	1.21	1.60
Mexican American	1.90	0.79

Data: Birthweight ≥ 500g; gestational age > 22wks; singletons; US residents

I next ran the SPF logistic regression models for all groups/time periods with new optima at race/ethnic, sex and time-specific points. I ran two sets of models, the first with race/ethnicity as a covariate and the second within separate race/ethnic groups, all by time period. Lastly, in order to make comparisons, I also included logistic regressions with more traditional birth outcome measures to see what picture we get with each approach. The parameters included were: low birthweight, preterm birth and the interaction of the two.

CHAPTER 3: RESULTS

3.1 Traditional Approaches

I will begin this chapter with a discussion of conventional approaches to birth outcome and infant mortality research. Tables 10 and 11 represent the most general (and the most frequent) approach to controlling for adverse birth outcomes within recent years. As discussed above at length, the history of birth outcome parameterization involves simple dichotomies that isolate the most adverse births in order to account for the link between pre- and postnatal variables on the health and well-being of children. Of more importance for this study is the study of race/ethnic differentials in these well-being measures. Again, it is imperative to note that standard measurement schemes have been primarily developed utilizing data that lacked any real race/ethnic diversity. As I move toward a more complex parameterization of birth outcomes, it behooves us to get a race/ethnic "picture" of the extent to which research on any given dataset will result in using traditional approaches. We can then move from this traditional picture to a new one using more advanced techniques and approaches.

Table 10 includes six sets of infant mortality models utilizing standard logistic regression over this study's comparative time period. In period one, I include three models. Model 0 presents Odds Ratios (ORs) for bivariate relationships between reference and study categories. Models 1 and 2 are

Table 10. Logistic Regression Analysis of Infant Mortality: **U.S. Females***

PERIOD	1989-1991			1995-1997		
MODEL	0	1	2	0	1	2
Race/Ethnicity						
NH-Black	2.23	1.25	1.26	2.04	1.23	1.24
Mexican American	1.04	1.02	1.03	1.01	1.01	1.01
Gestational Age						
Preterm	9.58	3.02	2.32	9.55	2.88	2.27
Birthweight						
Low Birthweight	16.46	8.43	6.91	16.55	8.62	7.28
Interaction						
PT*LBW	---	---	1.59	---	---	1.50
Intercept		-5.87	-5.83		-6.13	-6.09
-2LL		324196	323972		247625	247503

Notes – LBW: < 2,500g; Preterm: < 37 weeks gestation. Model 1 equals bivariate ORs.
*All odds significant at 0.01 level.

multivariate models that control for all presented variables. The same pattern is repeated for period two. Covariates include race/ethnicity, preterm, low birthweight and an interaction term for preterm and low birthweight. Beginning in period one, model 0, we see the Non-Hispanic Black (Black) disadvantage presented by bivariate ORs. Blacks experience over two times the odds of infant mortality as compared to Non-Hispanic Whites (Whites).

When controls are added in model 1 for adverse birth outcomes, there is a dramatic drop in the Black OR from 2.23 to 1.25. This 44% drop in the OR is indicative of the over two-fold disadvantage Blacks experience in the birth outcome distribution compared to Whites (cf. Table 4, Chapter 1). The addition of an interaction term for preterm and low birthweight does not substantially change

the Black OR in model 2. Moving to time period two, we see the exact same pattern emerge for Blacks. Of importance for future discussion is the third model in each time period. Using this traditional approach, we get the impression that the Black infant mortality disadvantage is strongly correlated to the maldistribution of Black births, not necessarily the outcome-specific risks for Blacks versus Whites. Also important to note then is that over time there has not been much improvement in this maldistribution nor in any outcome-specific mortality risk.

Of additional interest are the results for Mexican Americans (Mexican American). As is expected (given the near identical birth outcome distributions and associated infant mortality risks), Mexican American infants experience nearly identical mortality rates as compared to Whites over both time periods. The "Epidemiologic Paradox" is clearly shown.

The adverse birth outcome parameters give us important information as to their relationship to each other. It is clearly evident how substantively and statistical powerful these basic adverse birth outcome measures are. Infants born before 37 weeks gestation are clearly extremely disadvantaged. These preterm infants experience over nine times the odds death in the first year of life as compared to their non-preterm counterparts. Of even greater bivariate impact is the effect of being born under 2,500 grams. Low birthweight infants have over 16 times the odds of infant mortality as compared to their non-low birthweight

counterparts. Given the highly correlated nature of these two straightforward measures, Models 1 and 2 are important in that they include both variables simultaneously in the logistic regression model. With full controls, the effect of each adverse birth outcome is lessened by 76% for preterm and 58% for low birthweight. Even with these dramatic drops, these birth outcomes are highly predictive of infant mortality and morbidity. Given the differential percentage drop in the OR for each birth outcome, it would seem that low birthweight is the dominant statistical adverse birth outcome. The ratio of the OR between low birthweight and preterm changes from 75% to almost 200% when moving from model 0 to 2. Period two shows an even greater percentage increase in this ratio (from 73% to 221%). This again confirms the independent statistical relationship between these still-correlated dichotomous measures. A rarely used interaction term is included to ascertain some measure of the independence of these two variables. This interaction term does identify an additional 50-60% increase in the odds of infant death for infants that are doubly disadvantaged on these measures. There is confidence in both the data and the measures as all estimates are stable over time. A curiosity is the slight increase in the odds for low birthweight births across all models over time whereas the estimate of preterm and interaction effects drop slightly. For this and other reasons, analyzing race/ethnic-specific models may give clues as to whether trends presented here are indicative of all

Table 11. Logistic Regression Analysis of Infant Mortality: **U.S. Females***

TIME	1989-1991			1995-1997		
MODEL	NHW	NHB	MX	NHW	NHB	MX
Gestational Age						
Preterm	2.71	1.70	1.86	2.51	1.73	2.23
Birthweight						
Low Birthweight	8.69	3.88	8.64	8.75	4.08	9.75
Interaction						
PT*LBW	1.28	2.58	1.90	1.27	2.39	1.45
Intercept	-6.00	-5.39	-5.95	-6.23	-5.67	-6.26
-2LL	198024	96732	28639	149948	67057	30143

Notes – LBW: < 2,500g; Preterm: < 37 weeks gestation. Model 1 equals bivariate ORs.
Data – BWT > 500g; GA > 22wks; Singletons; US residents.
*All odds significant at 0.01 level.

groups or are a result of the influence of Whites given their demographic dominance.

Table 11 includes race/ethnic specific models with full controls for adverse birth outcomes for both time periods among female infants. These models allow from some preliminary analyses of the specific distributional and risk differences experienced by each race/ethnic group given standard birth outcomes measurement schemes. Scanning quickly across each outcome and race/ethnic groups over time, one distinct result stands out: the Black "advantage" within main effect adverse birth outcome parameters. For both periods, Black infants maintain a strong advantage in the odds of death versus the other groups. The most evident example is that of low birthweight, where Blacks experience less than half the odds of infant death compared to both Whites and Mexican

American. This occurs even after controlling for both preterm infants and the interaction term. The Black advantage is also evident, albeit not as strong, for preterm infants. Where there is not an advantage for Black infants is in the estimate of the interaction term. For this estimate, Blacks experience almost twice the odds of mortality. This is an important estimate as it points to some disparity between the groups when dealing with the most disadvantaged infants.

In Table 11, many of the same patterns emerge as in Table 10. Low birthweight overshadows the effect of preterm birth but does not eliminate it. The interaction odds decease over time for all groups. Of special interest is the finding that, even among separate groups, there is an increase in the odds of low birthweight death over time. The increase is especially marked among Mexican American infants, with their ORs increasing 13% (8.64 to 9.75). The same pattern for preterm infants does not hold. Whites experienced a 7% decrease in their preterm OR while Blacks experienced almost no change and Mexican American infants experience another main effect OR increase of 20%. Given that Mexican American infants also display the largest decrease in their interaction term (24%), it is possible that there is interplay between the decreasing odds for the most disadvantaged infants (those that are preterm and low birthweight) and the main effects for low birthweight infants generally and for both adverse birth outcome main effects among Mexican American infants.

In conclusion, this analysis utilizing a traditional approach to birth outcome parameterization results in two important conclusions for my purposes: 1) After controlling for adverse birth outcomes, the Black disadvantage in infant mortality is greatly reduced; and 2) within race/ethnic-specific models, there appears to be a Black advantage among main effect adverse birth outcomes. Given these conclusions, the next step is to apply a new approach to the same data and witness whether there is any significant change in the conclusions above.

Table 12. GAM-calculated Optimal Birthweight (z-score):
Female Infants

TIME	1989-1991	1995-1997
Gestational Age	39-41 weeks	39-41 weeks
Race/Ethnicity	Optimum	Optimum
NH-White	1.51	1.42
NH-Black	1.21	1.60
Mexican American	1.90	0.79

Data: Birthweight > 500g; gestational age > 22wks; singletons; US residents

3.2 Race/Ethnic Optima

Table 12 (reproduced from Table 9 above) displays the results from the GAM-assisted calculations on optimal birthweights by race/ethnicity and time for female infants. By optimal, I refer to the birthweight point estimate that exhibits the lowest risk of infant mortality between and including 39 and 41 weeks gestation. What is listed is this point estimate in standard deviations based upon the transformed z-score distribution set around its mean and standard deviation.

With a mean of zero and standard deviation of one, these transformed distributions are utilized in the creation of the birth outcome parameters created by SPF. A note of caution should be reiterated: these optima represent estimates that can easily vary when the gestational age range I utilized is varied in any way. Given that normal human gestation is 40 weeks, the range chosen would seem to capture the best starting point for analyzing optima.

In time period one, Whites experience their lowest infant mortality risk at 1.51 standard deviations above their mean. This is a birth of approximately 9.1 lbs. in standard form. For Blacks in time period one, the standard weight of the optimum is approximately 8.4 lbs. and for Mexican Americans it is approximately 9.5 lbs. What is interesting to note is that there seems to be small correlation between optima and infant mortality or other outcomes that might serve to gauge the validity of the optima calculated. Although Whites and Mexican Americans share similar risk levels, their optima vary widely from each other, reversing in rank order from period one to period two. As for Blacks, the highest mortality group, they experience neither the highest nor lowest optimum uniformly. In period one, they exhibit the lowest optimum, and in period two, the highest.

Over time comparisons also do not seem to shed much light on the meaning of these optima levels or changes. The optimum for Whites slightly increases over time by 6% while the optimum for Blacks increases by 32%. Mexican American infants show the largest change over time with their optimum

decreasing by a substantial 140%. It remains to be seen how these results will illuminate our understanding of the SPF model before investigating the results from those methods. It is to those results we now turn.

3.3 SPF models

3.3.1 Race/Ethnicity as Covariate: 1989-1991

Tables 13 through 17 display results utilizing the SPF approach coupled with my nonparametric regression (GAM)-calculated optimum selection method. These models allow for the standardization of race/ethnic birthweight and gestational age distributional differences. This standardization should allow for less-biased adverse birth outcome measures to be constructed. Table 13 begins with these results among female infants for time period 1989-1991. For comparative purposes, the GAM-assisted models are placed in the second set of columns alongside the OR estimates utilizing the SPF approach. The SPF models employ a +1 SD cutpoint for all gestational-age standardized birthweight data resulting in the S and H parameters. The E and L parameters represent gestational age deviations from 39 or 41 weeks gestation on a z-score scale. Model 0 does not adjust for any covariates and displays bivariate ORs only. Model 1 includes simultaneous controls for all variables estimates presented while Model 2 includes the interaction terms. By contrasting the estimates and infant mortality levels, we

can begin to make judgments concerning the validity and utility of this new approach for using individual-level data.

Beginning with Table 13, we notice an important first clue as to the validity of these models. As repeated from Model 0, Table 10, the bivariate odds for Blacks is 2.23. Using the SPF approach (again optimum set at +1 standard deviation above the race/ethnic-specific mean birthweight and gestational age) in Model 1, the period one bivariate odds of Black infant death versus Whites falls slightly to 2.13. Comparing this number to the corresponding one in Table 1 of 1.25 (main effects only), one can immediately see how standardizing for each race/ethnic group's own birthweight and gestational age distributions has resulted in an OR that is extremely close to the bivariate one of 2.23. Within the full model with interaction terms (Model 2) we see little change in the Black OR of 2.13 (as compared to 1.26 in Model 2 of Table 10). The SPF model results in widely different ORs for Blacks as compared to Whites with Blacks maintaining their more than double odds of infant mortality regardless of controls for adverse birth outcomes.

Table 13. Logistic Regression Analysis of Infant Mortality: **U.S. Females (1989-1991)**

OPTIMUM	SPF (SD)			GAM (SD)		
MODEL	0	1	2	0	1	2
Race/Ethnicity						
NH-Black	2.23	2.15	2.13	2.23	2.69	2.54
Mexican American	1.04	1.03	1.02	1.04	0.76	0.80
Gestational Age						
Early Birth (E)	2.34	2.41	2.02	2.34	2.41	1.97
Late Birth (L)	1.40	1.53	1.51	1.40	1.53	1.44
Birthweight						
Small Weight (S)	2.04	2.28	1.99	1.87	2.23	1.88
Heavy Weight (H)	1.24	1.11	1.81	1.61	1.42	1.88
Interaction						
E*S			1.14			1.15
E*H			0.85			0.92
L*S			1.03†			1.05
L*H			0.93†			1.13
Intercept		-7.23	-7.05		-7.61	-7.28
AIC		299,252	297,895		299,351	298,177
-2LL		299,238	297,873		299,337	298,155

NOTE: Unless otherwise noted, all odds ratios significant at 0.01 level; *$p \leq 0.05$; †$p > 0.05$

When we move to the models that allow for different optima among the groups, we see another increase in the OR for Blacks. Blacks experience an OR of 2.69 in the main effects models and 2.54 when interaction terms are added. The full model (Model 2) represents a 100% increase in the odds of infant mortality as compared to the traditional approaches in Model 2, Table 10. Again, utilizing the SPF model with GAM-assisted optimum calculation results in substantially higher

Black ORs both from the bivariate and traditional full models. Looking at the intercept and AIC values for both sets of SPF models show that these models isolate a "normal" population with quite lower infant mortality rates as well as much lower AIC values (representing models with better fit). The intercept for the traditional approach results in an infant mortality rate of approximately 29.4 infants per 10,000 while the SPF models (+1SD and GAM-calculated SD) results in 8.7 and 6.9 infant deaths per 10,000 live births respectively (a 70% and 77% decrease in infant mortality).

Moving from SPF to GAM models, there is a different trend in the Black OR that warrants mentioning. Within SPF models, the move from bivariate to full models results in gradual decreases of the Black OR while the GAM models result in increases from the bivariate OR when adverse birth outcomes are controlled. This signifies the first piece of evidence that even when standardized, Blacks may still maintain lower odds of infant mortality among some set of adverse birth outcomes. Of special interest is the increase in the Black OR within the GAM models from a full model without interaction terms to one including them. This again may be a slight indication of Black advantage among a set of interaction outcomes relative to Whites. Race/ethnic-specific models presented later in this chapter will allow for a more thorough investigation of this possibility.

Mexican American infants still exhibit OR similarity with White infants in both sets of models. When using the GAM approach, Mexican American infants show some gains in their overall OR when full controls are added. These data again point to the relative uniformity between White and Mexican American infant mortality experiences that will be more fully explored when analyzing race/ethnic-specific models below.

For the remainder of the parameter estimates, there is clear evidence as to the overall power of these models in allowing for non-linear infant mortality risk to be effectively modeled with categorical variables and logistic regression. For example, infants born "E" or "Early" (premature) by one standard deviation from their race/ethnic and sex-specific mean experience a two-fold plus increase in their odds of infant death relative to those infants born between 39 and 41 weeks of gestation. When controls for birthweight status ("Small" or "S" and "Heavy" or "H") are added, the Early OR remains close to two for both the SPF model (OR=2.02) and GAM model (OR=1.97). A drop from an OR well above two to one near two occurs when interaction terms are entered in the model. This result is to be expected, given the voluminous amounts of data and research on the effects of prematurity on infant mortality coupled with the additional mortality risk associated with infants that are also disadvantaged in regard to birthweight (Early and Small, Late and Heavy and Late and Small) and are somewhat advantaged when prematurity is coupled with better than optimal birthweight

(Early and Heavy) (cf. Powers et al. 2006). Research has also pointed to the deleterious main effect of being born post-term and that result is borne out here. Utilizing both models, infants born one standard deviation above their group mean for gestational age experience an over 40% increase in their odds of death. Even after controlling for the growth of infants in terms of birthweight, there still exists some negative effect of remaining *in utero* past the biological gestational norm for humans.

This strong pattern of adverse birth outcome on both sides of the optima is also evident among infants born either Small or Heavy. At the outset, the bivariate ORs differ, especially for the H parameter. This should not be surprising given that the overall average optima for all three groups in this time period is approximately half a SD higher than for the SPF model of +1 SD. This does result in the H term being composed of slightly higher-rsik births, thus the difference between the SPF Model 0 H OR of 1.24 versus 1.61 for the GAM Model 0. Surprisingly, the ORs for both Small and Heavy births are almost identical when full controls are added that include interaction terms. The GAM model results in a higher main effect for Heavy infants than does the SPF model (1.42 versus 1.11) when interaction terms are excluded. This may indicate the utility of race/ethnic-specific optimums. In this case, these variable optima allow for more precise identification of infant disadvantage among those born above their age-specific mean. In Model 2, for both modeling approaches, the H term

increases dramatically to virtually equal that of the S term. Here, the interaction terms allow for differential effects of age and weight interactions. This further establishes the importance of being born as close as possible to gestational age-specific optima. Any deviations from these optima result in increased risk of death. I also note in passing that another explanation does present itself for the high H estimate. As will be discussed further in the following chapter, the choice of the optima may also artificially inflate the H estimate by misidentifying H infants when there is in reality a linear association between weight gain and survivability at certain gestational ages. Suffice it to say that this is an important issue and one that is explored more in detail later.

Of further importance is the independent relationship between gestational age and birthweight estimates as shown in the interaction term results. Being Heavy and Late confers additional disadvantage as seen through the positive estimates for their interaction term. Infants born both Early and Small experience a double disadvantage as well, with a 14% then 15% increase in their odds of death from the SPF to the GAM model. This interaction term is the largest one estimated among all interaction terms and fits well with the logical and experiential conclusions on the negative effect of premaurity and small-for-gestational-age status. Infants born both Late and Heavy experience positive or negative odds of mortality depending on the model utilized. The SPF model shows an OR below unity at 0.92 while the GAM model shows an increase in the

odds of mortality of 13%. Here we find additional evidence of the divergence of the two models and the importance of optimum selection in the estimation of parameters. Given the 15% decrease in the GAM model intercept versus the SPF model, indicating a more precise estimate of the most "optimal" cross-classification of infants, one might want to tentatively conclude in favor of the superiority of the GAM-calculated variable optimums. In the full model the AIC and -2LL decreases from the SPF to the GAM models may also bolster that conclusion. For now, I will leave that assessment until the Discussion chapter of this dissertation. For now, note that the interaction estimates that denote both Early and Heavy births confer some small advantage (OR=0.85 and 0.92, respectively). This result is what one would expect given what has been published concerning positive intrauterine weight gain irrespective of gestational age. Lastly, there is a small additional disadvantage of being born Late and Heavy. With full controls, one might conclude that independent effects of gestational age and birthweight are multipliers in conferring mortality disadvantage. Given the small ORs for these estimates (1.03 and 1.05 respectively) it remains to be seen whether this interaction really increments the large odds of mortality for the main effects of Late and Heavy births.

3.3.2 Race/Ethnicity as Covariate: 1995-1997

Table 14 details these results for period two. What stands out at the outset is the Black main effect estimate for the full GAM model in Model 2. Blacks

experience an OR of 1.80 in this model. The corresponding model OR for period one (GAM, Model 2, Table 13) is 2.54. These results are again stark in comparison with traditional approaches that display a much closer gap between White and Black infants when adverse birth outcomes are controlled for. Conversely, the period two, Model 2, GAM-Black estimate in Table 14 is 29% lower than the parallel estimate in period one (Table 13). Lastly, there is much more uniformity to the estimates for Black infants within the SPF models over both time periods. Both the levels and trends among SPF Black estimates are strikingly similar (ORs begin slightly over two and fall slightly). Here, the GAM-calculated estimates may have played a significant role in these results, resulting in less precise estimates. It is also possible that changes in the outcome-specific mortality rate reductions are also working to lower this estimate. In either case, for time period two there is less clear evidence of the supremacy of the GAM model for estimates of Black infant mortality.

Table 14. Logistic Regression Analysis of Infant Mortality: **U.S. Females (1995-1997)**

OPTIMUM	SPF (SD)			GAM (SD)		
MODEL	0	1	2	0	1	2
Race/Ethnicity						
NH-Black	2.04	2.03	2.03	2.04	1.77	1.80
Mexican American	1.01	1.02	1.01	1.01	1.62	1.49
Gestational Age						
Early Birth (E)	2.23	2.34	2.11	2.23	2.34	2.07
Late Birth (L)	1.46	1.55	1.51	1.46	1.55	1.44
Birthweight						
Small Weight (S)	2.02	2.29	2.11	1.99	2.23	2.00
Heavy Weight (H)	1.29	1.14	1.82	1.57	1.32	1.83
Interaction						
E*S			1.08			1.09
E*H			0.87			0.93
L*S			1.02†			1.05
L*H			1.05†			1.18
Intercept		-7.51	-7.42		-7.68	-7.50
AIC		228,566	228,007		228,696	228,244
-2LL		228,552	227,988		228,682	228,222

NOTE: Unless otherwise noted, all odds significant at 0.01 level; *$p \leq 0.05$; †$p > 0.05$

The results for Mexican American infants are tenuous as well. Although the SPF model estimates for this group are almost identical over time (1.02 and 1.01 respectively for SPF Model 2 in Tables 13 and 14), in period two, GAM, Model 2, there is a sharp increase in their OR (from 0.80 in period one, Table 13 to 1.49 in Table 14). This advantage turned disadvantage is most likely due to the small numbers of Mexican American deaths within this study. There are roughly

66% to 75% less Mexican American deaths compared to Blacks and far less as compared to Whites. For this data-intensive analysis, small numbers of deaths may result in unstable optima calculations. In this case, it is possible that the ratio of birth and deaths between Whites and Mexican American within the range of normal gestational ages (39 to 41 weeks) may have fallen below some statistical threshold resulting in the change in optima for females from almost two to below one standard deviation over the study time periods. It is far more likely that the optima in time two should be roughly equivalent to time one given the relatively short study period and our current understanding of the difficulty in shifting birthweight distributions and outcome-specific risks. With the exception of pulmonary surfactant therapy (1990 onward) to reduce RDS deaths or the back-to-sleep initiative to reduce SIDS deaths (1992-1994), I can think of no other major medical intervention that would have drastically increased the viability of the low-end birthweights of normal gestation for Mexican American infants *only*, thereby decreasing the optimum almost one whole standard deviation. As noted, given the variability in optima changes for both Whites (decrease) and Blacks (increase), it remains to be seen how one might accurately relate optima shifts to conceptually and methodologically useful conclusions. A more thorough discussion of this issue will be addressed in the concluding chapter of this study.

As for the adverse birth outcome parameter estimates, there is a strong pattern of corresponding results from period one to period two with many

estimates being nearly equal in value. Of special note is the finding that in both the SPF and GAM models with full interactions (Model 2), both the main effect estimates for traditionally adverse outcomes of E and S have increased in value over time while their interaction term (E*S) has decreased ever so slightly. This may point to positive changes in the efficacy of Neonatal Intensive Care Units (NICU) treatments exclusive to the most disadvantaged infants (possibly through Medicaid) or some artifact of the variant optima-scheme. The latter issue may be salient inasmuch as the model fit statistics result in nearly equal infant mortality levels among the normal reference population.

3.3.3 Race/Ethnicity-Specific Models: Non-Hispanic Whites

Table 15 looks into race/ethnic-specific models in order to assess these new modeling strategies and their estimates on the Black advantage despite adverse outcome births. Beginning with White female infants, some important trends can be identified. Although infant mortality rates for "optimal" births have indeed fallen (given the decreasing intercept over time), there is variability in the trends of each birth outcome effect. As discussed earlier, of note is the similar optimum found among Whites using the GAM approach over both time periods (1.51 and 1.42 respectively). This is heartening in that it helps to confirm the veracity of this finding and approach, insofar as White females are concerned. Given this result, we may be confident in moving forward with parameter estimates interpretation for both time periods. First, the similarity in the bivariate

Table 15. Logistic Regression Analysis of Infant Mortality: **NH-White Females**

PERIOD	1989-1991			1995-1997		
OPTIMUM	(1.51)			(1.42)		
MODEL	0	1	2	0	1	2
Gestational Age						
Early Birth (E)	2.29	2.35	2.05	2.23	2.29	2.11
Late Birth (L)	1.45	1.46	1.46	1.48	1.48	1.50
Birthweight						
Small Weight (S)	2.02	2.25	2.02	2.03	2.28	2.13
Heavy Weight (H)	1.78	1.60	2.09	1.74	1.42	2.07
E*S			1.10			1.06
E*H			0.92			0.91
L*S			1.01†			1.00†
L*H			1.09†			1.17*
Intercept		-7.81	-7.61		-8.02	-7.90
AIC		184,284	183,828		139,162	138,942
-2LL		184,274	183,810		139,152	138,924

NOTE: Unless otherwise notes, all odds significant at 0.01 level; *p≤0.05; †p>0.05

odds across both time periods found in Model 0 is interesting. This may portend very little change in their odds levels as we move forward. When controlling for main effects only (Model 0 to Model 1), subtle changes and shifts occur.

For both time periods, E estimates increase slightly, L estimates remain similar, S estimates increase appreciably while H estimates decrease likewise. Of these constant trends, the most telling is the increase in the S estimate with very little change in the E estimate. When both variables are controlled simultaneously, it would seem that against the age-specific optimal birthweight population, Small infants are more disadvantaged when gestational age is taken into account. Given

that almost half of the deaths for any given population occur within the optimal gestational age span, when a control is added for gestational age, a separation occurs between the wider variance in lower mortality risk among later ages versus the smaller variance in risk when deviations occur from the means at much lower gestational ages. In Model 1, normal gestational age births have a value of zero, thereby relegating only the "deviants" to the analysis. The addition of interaction terms (Model 2) serves to reduce the main effect for S almost back to the bivariate level.

The same trend occurs for the E estimate, but with some interesting differences. In period one, the unadjusted (Model 0) E OR (2.29) is very near the main effects-only level of Model 1 (OR=2.35). The same trend occurs in period two (2.23 and 2.29, respectively). Like those infants born Small, isolating those births with a zero value on the birthweight estimate allows for a slight increase in the E estimate. In addition, the E estimates decreases in Model 2 to well below the bivariate odds, indicating the importance of the interaction terms included in Model 2 across both time periods.

For the rest of the main effects, the trend is mixed when moving from Model 1 to Model 2 across time periods. Late births show almost identical odds across all models and time periods while Heavy infant estimates decrease when including only gestational age main effects (1.78→1.60 and 1.74→1.42, respectively). This latter result is logically consistent as the deletion of the

optimal age group allows for less deleterious effects of heavy weight at all other gestational ages, especially young ones. Although there may indeed exist optimums at many younger gestational ages, previous research would indicate that any positive birthweight accumulation at early ages is beneficial as compared to the same pattern at term and post-term ages. This hypothesis is further bolstered by Model 2 H estimates. Moving from Model 1 to Model 2 for both time periods, H estimates increase 31% and 46%, respectively. Both these increases in the H ORs in Model 2 are the result of adding interaction terms. Specifically, the E*H and L*H parameters tell the story. These interaction terms signify the isolation of two specific infant populations with opposite mortality experiences. Those infants born both Early and Heavy are slightly advantaged relative to those born either Early or Heavy. Here again, we have evidence of an inverse relationship between birthweight and infant mortality risk as gestational age decreases. On the other side of the equation is the effect of being born Late and Heavy results in a slight disadvantage. The increase in the odds over time among L*H infants may reflect real increases in macrosomic mortality rates. Controlling for these interactions allows the main effect of Heavy to reflect the "true", overall risk of being born above one's race-ethnic optimum at optimal gestational ages only. The trends for the other interaction terms are sensible and straightforward. The adverse interaction term of E*S are above unity as expected.

The reduction in the E*S term over time may indicate advances in NICU treatments even during this short time span.

3.3.4 Race/Ethnicity-Specific Models: Non-Hispanic Blacks

Table 16 displays results for Black females over time. In summary, almost all the trends found among Whites also are found here. The main focus and utility of this analysis will be comparing the estimate levels against those for Whites. It is important to analyze whether Black estimates are truly lower than those of their White counterparts in an effort to answer the question of Black advantage in standardized adverse birth outcomes. As mentioned in the discussion

Table 16. Logistic Regression Analysis of Infant Mortality: **NH-Black Females**

PERIOD	1989-1991			1995-1997		
OPTIMUM	(1.21)			(1.60)		
MODEL	0	1	2	0	1	2
Gestational Age						
Early Birth (E)	2.57	2.63	1.90	2.46	2.51	2.01
Late Birth (L)	1.78	1.80	1.60	1.79	1.80	1.34
Birthweight						
Small Weight (S)	1.91	2.14	1.59	1.80	2.02	1.63
Heavy Weight (H)	1.20	1.06†	1.71	1.55	1.30	1.75
E*S			1.25			1.17
E*H			0.86			0.95*
L*S			1.10*			1.21
L*H			0.96†			1.18†
Intercept		-6.97	-6.47		-7.41	-6.96
AIC		87,899	87,164		61,132	60,927
-2LL		87,889	87,146		61,122	60,909

NOTE: Unless otherwise notes, all odds significant at 0.01 level; *p≤0.05; †p>0.05

of Table 5, the optima for Black female infants are somewhat constant with only a .4 standard deviation increase in the optimum over time. This would amount to a birthweight standard deviation of roughly 200 grams or 7 ounces.

3.3.5 Race/Ethnicity-Specific Models: Mexican American Females

Table 17 displays the results over time for Mexican American infants. The optimum for gestational ages 39 to 41 weeks in period one is 1.9 standard deviations above their mean birthweight. Within period one, bivariate ORs (Model 0) show similar patterns to the other race/ethnic groups discussed with Early and Small parameter estimates above two. Interestingly, the highest

bivariate OR among this group is the Heavy parameter at 2.36. When main effects are both entered into Model 1, there is a slight increase in the S estimate from 2.17 to 2.35 and a much larger increase for the H parameter (2.36 to 2.69). Both E and L remain virtually unchanged.

This result points to a relatively higher mortality average for H infants born outside of the 39 to 41 gestational age range. I suspect that this slight increase is due to higher risks associated with infants born after the 41st week more so than premature infants. Model 2 allows a confirmation of this possibility.

Table 17. Logistic Regression Analysis of Infant Mortality: **Mex-American Females**

PERIOD	1989-1991			1995-1997		
OPTIMUM	(1.90)			(0.79)		
MODEL	0	1	2	0	1	2
Gestational Age						
Early Birth (E)	2.30	2.37	1.86	2.28	2.34	2.11
Late Birth (L)	1.51	1.52	1.27*	1.55	1.56	1.59
Birthweight						
Small Weight (S)	2.17	2.35	1.92	2.26	2.53	2.33
Heavy Weight (H)	2.36	2.69	2.42	1.38	1.35	1.92
E*S			1.17			1.08
E*H			0.99†			0.88
L*S			1.12*			1.01†
L*H			1.40*			0.96†
Intercept		-8.23	-7.74		-7.75	-7.67
AIC		26,860	26,756		28,162	28,079
-2LL		26,850	26,738		28,152	28,061

NOTE: Unless otherwise notes, all odds significant at 0.01 level; *p≤0.05; †p>0.05

If correct, controlling for interaction effects should lower the main effect for H and result in an OR above unity for the L*H estimate. This is indeed the case, with the H estimate falling to 2.42 and the interaction term for L*H equaling a 40% increase in the odds of infant mortality over and above being either Late or Heavy. Model 2 shows reductions in all main effects as expected given the inclusion of interactions. Of note is the 62% increase in the infant mortality rate for the reference population as shown by the increasing intercept term from Model 1 to Model 2, period one. Although not a measure of model fit per se, Mexican Americans are the only group to exhibit an increase in the infant mortality rate when moving to a fuller model. Even so, the AIC and -2LL values do decrease for both time periods, indicating some measure of support for a better fit of the data in Model 2 than in Model 1.

With the large decrease in the optimum from period one to period two (58%), one wonders what that will mean for the validity of the model. Looking at the bivariate estimates in Model 0, we do indeed find one large difference in the ORs. Specifically, the only estimate that changes in any great measure is the bivariate odds for Heavy infants. Between period one and period two, the OR decreases from 2.36 to 1.38. This result clearly shows how the changing optima can alter the estimates in the several models. Of course, this is to be expected. As the optimum moves, higher risk births "move" from one category to another. Model 1, with controls for main effects added, displays almost the exact same

estimates from Model 0. The addition of interaction terms reverses the trend seen in period one, namely the H parameter increase. This is the trend seen in all other periods among Whites and Blacks. The only visible difference is the L*H estimate, an estimate below unity (OR=0.96). This runs counter to the research that indicates a deleterious effect in being born both post-term (in this case 42 weeks or greater) and above the optimum age-adjusted birthweight.

3.3.6 Race/Ethnicity-Specific Models: Race/Ethnic Comparisons

When looking at both White/Black Model 0 and Model 1 comparisons, a consistent pattern emerges. For both bivariate and main effects, Blacks experience higher odds for E and L parameters while experiencing lower odds for S and H estimates. When only main effects are controlled for, it would seem that Blacks have a differential birth outcome risk experience as compared to Whites. Black infants with deviations on either side of 39 or 41 weeks gestation experience higher risk than their White counterparts (the ratio of the ORs for E and L range from 110% to 123% over both time periods). On the other hand, controlling for gestational age results in lower ORs for Black birthweight deviations as compared to Whites (a ratio range from 66% to 95%). Even after standardizing for each race/ethnicity's specific birth distribution, results show a significant birthweight advantage at below-optimal weights for Blacks. This advantage is reversed for age deviations. Again, the idea that Black infants are more robust at larger deviations from their optimal weight is confirmed here. The

addition of interaction terms in Model 2 does change the relationship. For the three out of the four E/L parameters, Black infants now display a slight advantage in their ORs versus White infants (from 89% to 95% advantage) while the only Black disadvantage occurs for the L estimate for period one (110% ratio of the odds). For birthweight deviations, the Black advantage remains in all time periods for both directions of deviations. In period two for example, Blacks are at a 77% advantage among S infants who are one standard deviation from the age-specific mean while L infants are at an 89% advantage. One estimate where there seems to be a distinct Black disadvantage is in the odds of mortality among E*S infants. This combination of adverse birth outcomes increases the main effect of each separate estimate by 1.25 the odds in period one and 1.17 the odds in period two. These ORs are higher than the White infants ORs by 14% and 10% respectively. So for the most disadvantaged infants, Blacks experience lower survivability than Whites within their own distributions.

In relation to Black and Whites, Mexican American infants experience very similar patterns both within models and over time. Like the relationship between Blacks and Whites, Mexican Americans exhibit higher odds of both Small and Heavy estimates as compared to Blacks. The Black advantage associated with these adverse outcomes over Whites is also maintained for Mexican American infants. This is additional evidence of the "Epidemiologic Paradox" so omnipresent in research and yet still unclear etiologically.

CHAPTER 4: DISCUSSION

4.1 Optimum Measurement

4.1.1 Optimum Measurement Advances

In this dissertation, I have endeavored to advance the knowledge and understanding of the complex relationship between birth outcomes and infant mortality utilizing recent improvements in both methodological approaches and statistical methods. The use of infant mortality as an outcome variable serves to place birthweight and gestational age within the causal pathway between important socioeconomic, sociodemographic and biological/physiological contexts, namely those of maternal health and infant health (cf. Hummer et al. 1999; Wise 2003). By applying the model created by SPF and expanded on by others (Powers et al. 2006) to three different race/ethnic groups over two time periods, I have attempted to investigate the efficacy of this new modeling strategy. In addition, I have utilized a more recent statistical approach (nonparametric regression) in order to increase the accuracy of the SPF model by statistically identifying optimal "zones" of infant mortality risk and model deviations from these zones therein. However, no effort of this type culminates in perfect answers to perfect questions. Invariably, several results of this project have led to further questions on both the efficacy of approaches used as well as conceptual assumptions brought to this work. In this final chapter I summarize

and contextualize the important findings as well as elucidate any advantages of this approach in and any questions, problems, issues and caveats that arose given my results. Lastly, I will be critical to place this work within the context of the larger sociological and public health concerns and issues that motivated me to undertake this project.

The first main finding of importance was the ability to statistically analyze the birthweight distributions in order to find an optimal birthweight point estimate from which infant mortality risk changes in either positive or negative directions. Whereas SPF systematically chose their optimum based upon visual inspection of the birthweight curves at 40 weeks gestation, my methodology utilized nonparametric regression GAM models to produce a fully-nonparametric regression equation with accompanying predicted values that closely approximated the actual data. This ability to move away from any parametric assumptions on the shape of the birthweight-risk curve allowed for the calculation of non-linear effects across the range of data points. As many have asked the question, beginning with Wilcox and Skeorven in 1992 and most prominently ending with Powers et al. (2006), the creation of some methodology to accurately identify the "true" shape of this relationship is critical in hypotheses regarding the role of birthweight in the causal chain as highlighted by extant debates (cf Wilcox 2003; Hertz-Piccioto 2003; Wise 2003). Given the variable optima calculated for each race/ethnic group over time, there is strong evidence that these models more

accurately represent critical *in utero* development. On the other hand, the ultimate focus is on the survivability of live born infants.

4.1.2 Optimum Measurement Limitations and Caveats

A major limitation of this search for optima was the inability to identify optima at every gestational age. Tables 18 and 19 list the gestational age-specific optima that were calculated using GAM models over time. Bold figures indicate gestational ages where u-shaped optima were not identified (GAM regression equation reduced to logistic regression equation). Results (Appendix 1) were calculated using these optima from this table in order to see what the effect would be on the SPF model parameter estimates. The results indicated that certain estimates suffer from this approach. For example, in Table A1, White females had an OR for Small infants of 1.1. Given the inverse relationship between gestational age-specific mean birthweight and risk of death, this result seems implausible over the full range of gestational age data. One possible explanation involves the construction of the S and H parameters. At any one gestational age, the optimum dictates whether a case above or below said optimum gets a positive value for either S or H variables. In the case of White females in period one, many early gestational ages exhibit no true optimum. In these cases, it stands to reason that any weight gain is salubrious. But for the important gestational ages of "normal" range (i.e. 39, 40 and 41 weeks) the GAM procedure does not result in any optima values below the maximum birthweight represented in the data for that gestational

age. Hence, within these gestational ages representing a large proportionate number of births, all births are characterized as being Small ("<0" value for S and "0" value for H). It is probable that the extremely small infant mortality risk within these ages overshadows the effects of being Small at other gestational ages. In order to create a baseline set of results for future comparison, the decision to combine weeks 39 through 41 and recalculate the GAM optima was made (reproducing the method of SPF in the process).

This combination did provide statistically stable optima that were reported and used as proxies for other gestational ages. Even so, the change in optima for Mexican Americans from +1.90 SD in period one to +0.79SD in period two provides further reason for caution in interpretations of both models with race/ethnicity as covariates and those models that are race/ethnic-specific. Future research should replicate this approach after creating protocols for the calculation of age-specific GAM optima. One possible method would be to utilize a "running average" approach in order to gain enough cases from neighboring gestational ages and create stable GAM-calculated optima. For example, the optima within this dissertation would represent the value for the 40^{th} week. Combining weeks 40 through 42 would give the value for week 41 and so on. This might be a fruitful protocol for increasing the precision of this parameterization scheme and better modeling of extant data.

Table 18. GAM Predicted Optima: Z-Score Birthweight on logit(IMR)

	1989-1991					
	NH-WHITE		**NH-BLACK**		**MEX AM**	
	OPTIMA	IMR	OPTIMA	IMR	OPTIMA	IMR
GA	x	ŷ	x	ŷ	x	ŷ
22	1.72	-1.98	2.19	-3.02	**3.21**	**-5.00**
23	2.26	-1.90	2.38	-2.83	**3.81**	**-12.90**
24	**3.29**	**-3.88**	**2.69**	**-5.23**	**2.92**	**-5.45**
25	**3.21**	**-4.69**	**2.38**	**-5.25**	1.38	-3.65
26	**2.86**	**-4.48**	**2.39**	**-4.85**	**2.38**	**-4.84**
27	**2.78**	**-5.62**	**2.30**	**-5.20**	**2.29**	**-5.07**
28	**2.42**	**-5.04**	**2.49**	**-4.24**	**1.99**	**-6.77**
29	**2.32**	**-5.31**	**2.39**	**-5.92**	**1.88**	**-14.00**
30	**2.18**	**-6.33**	**2.26**	**-5.58**	**1.88**	**-9.00**
31	**2.14**	**-5.85**	1.30	-5.16	**1.85**	**-11.70**
32	6.82	-5.26	1.91	-5.33	**2.93**	**-7.59**
33	**4.79**	**-7.48**	3.07	-5.75	**3.45**	**-6.80**
34	**8.28**	**-11.40**	2.01	-5.65	1.41	-5.81
35	**8.77**	**-6.93**	1.44	-5.36	**4.68**	**-10.50**
36	**9.37**	**-13.00**	1.68	-5.29	**5.21**	**-8.26**
37	0.97	-5.95	1.71	-5.75	1.15	-6.20
38	**10.18**	**-14.00**	2.85	-5.71	1.14	-6.15
39	**9.91**	**-13.10**	1.14	-5.75	1.33	-6.71
40	**10.51**	**-12.40**	1.58	-5.87	**6.58**	**-10.60**
41	**9.27**	**-12.30**	3.46	-6.18	2.00	-6.54
42	1.36	-6.42	3.27	-5.67	**5.42**	**-8.33**
43	1.81	-6.25	1.73	-5.41	**5.10**	**-7.16**
44	**5.73**	**-9.28**	1.56	-5.94	**4.52**	**-13.20**
45	1.09	-6.73	0.08	-5.74	**4.26**	**-10.30**
46	**4.96**	**-7.49**	1.85	-5.54	**3.67**	**-6.61**
47	**4.24**	**-9.90**	3.76	-7.78	0.25	-6.65
39-41	1.51	-6.62	1.21	-5.77	1.90	-6.73

Table 19. GAM Predicted Optima: Z-Score Birthweight on logit(IMR)

	1995-1997					
	NH-WHITE		**NH-BLACK**		**MEX AM**	
	OPTIMA	IMR	OPTIMA	IMR	OPTIMA	IMR
GA	x	ŷ	x	ŷ	x	ŷ
22	2.62	-2.36	1.25	-2.19	**3.29**	**-1.75**
23	**4.71**	**-2.54**	2.34	-2.42	1.85	-1.41
24	**3.34**	**-4.09**	**3.10**	**-5.56**	**2.96**	**-9.10**
25	**3.27**	**-3.67**	**2.98**	**-5.03**	**2.84**	**-9.40**
26	**3.10**	**-3.63**	**2.73**	**-3.78**	1.05	-3.43
27	**2.99**	**-4.60**	**2.69**	**-4.44**	**2.74**	**-4.27**
28	**2.57**	**-5.57**	1.48	-4.75	**2.21**	**-9.90**
29	**2.36**	**-6.88**	**2.52**	**-8.03**	**2.04**	**-6.66**
30	**2.23**	**-6.29**	1.12	-4.85	**1.93**	**-10.60**
31	**2.25**	**-5.70**	**2.40**	**-5.74**	**1.90**	**-5.25**
32	**6.36**	**-8.96**	**3.54**	**-5.56**	**3.56**	**-7.77**
33	**5.05**	**-5.93**	**3.85**	**-7.51**	**4.21**	**-12.40**
34	1.36	-5.50	1.00	-5.45	**1.12**	**-4.96**
35	1.71	-6.22	2.28	-5.81	**8.31**	**-12.50**
36	2.48	-6.14	**6.71**	**-10.70**	**6.63**	**-14.60**
37	**6.75**	**-11.60**	**9.81**	**-12.50**	0.58	-6.16
38	**10.35**	**-27.90**	**10.11**	**-10.60**	3.36	-6.63
39	**9.92**	**-13.70**	**10.67**	**-7.46**	1.39	-6.68
40	**10.35**	**-13.00**	**9.42**	**-7.27**	0.83	-6.90
41	0.96	-6.80	2.50	-6.50	0.66	-7.14
42	2.74	-7.02	0.79	-6.13	0.83	-6.46
43	**6.00**	**-10.80**	**8.29**	**-8.37**	**6.19**	**-16.00**
44	**9.00**	**-10.10**	1.21	-6.88	**5.23**	**-9.87**
45	**5.96**	**-9.38**	0.80	-6.51	1.00	-7.34
46	1.04	-6.51	0.27	-5.77	**6.16**	**-15.80**
47	**3.71**	**-9.57**	**3.61**	**-39.40**	**5.44**	**-2.27**
39-41	1.42	-6.82	1.60	-6.03	0.79	-6.87

Given the impressive results presented by Powers et al. (2006), two important methodological issues warrant mentioning. The first involves their utilization of infant day of death within their SPF modeling strategy. This novel addition is key to refining both the model and the use of available data. By focusing on day of death, the effect of sociodemographic and socioeconomic factors on infant mortality might be lessened. Using this modeling approach, Powers et al. (2006) find that, within the first day of death, the Black/White relative risk (controlling for SPF birth outcomes) is near unity while the relative risk for infants who die in the postneonatal period (28-364 days) is 2.673. This result alone is compelling evidence supporting the use of day of death in further analyses. Although this study utilizes logistic regression, while Powers et al. utilize proportional hazard models, the methodological improvement is evident within their results.

The second issue that Powers et al. bring to the table is the quasi-replication of my results within theirs. Although the statistical model is not identical, there are markers that provide important information. For example, within race/ethnic-specific models (Table 3) that do not control for day of death (Model 1), they find that White infants have relative risks for S/H parameters of 2.266 and 1.842 respectively while Black infants have S/H relative risks of 1.813 and 1.627. Compare these figures to the results from Tables 15 and 16, period two. For Whites the S/H ORs are 2.13 and 2.07 while for Blacks they are 1.63 and

1.75 respectively. From these numbers, it would seem that my approach results in smaller estimates for S infants (2.266 vs. 2.13 and 1.813 vs. 1.63) and larger estimates for H infants (1.842 vs. 2.07 and 1.627 vs. 1.75). What these results may point to is the real and important effect of optima selection. SPF birth outcome estimates for Whites and Blacks within this study use optima much higher than the 1 standard deviation optimum Powers et al. utilized (1.42 for Whites and 1.60 for Blacks). Since births are normally distributed within any given gestational age but deaths are highest at the extremes, it stands to reason that movement of the optimum up or down will result in meaningful changes in the parameter estimates, all things being equal. In this comparison, the optima are higher within this study. If this issue is salient, then we would expect as an optimum moves higher within a gestational age, there is a disproportionate decrease in the denominator (births) versus the numerator (deaths) within infant mortality statistical calculations of any type. This is the case here. The optima in this study are higher. We would expect both an *increasing* H estimate as a smaller number of H births are compared against a relatively unchanged number of deaths (at the upper end of the birthweight distribution) and a *decreasing* S estimate for opposite reasons (disproportionate addition of S births relative to S deaths found primarily at the lower end of the gestational age-specific birthweight distribution). This discussion highlights the importance of continued research into the most objective, efficient and valid estimation of race/ethnic-specific optima.

One additional approach would be to utilize the full power of GAMs and model the data in its full continuous form. This approach requires substantial computing power that is now within the realm of widespread accessibility. Further refinement of the approach presented here is possible and advisable. In an attempt to better capture the weight-risk relationship for an outcome that is relatively rare, modifications to the parameterization of the S and H parameters can be undertaken. One such modification would be to create groups of standard deviations in order to create larger bins of infant deaths for comparative analysis. For example, instead of an optimum for a group at exactly 1.5 standard deviations above the age-specific mean, that range might be widened to include neighboring birthweight z-scores (e.g. an optimum of 1.25 to 1.75 standard deviations). Graphical analyses may help display how "flat" the optimum really is. Statistical protocols can be developed that could give direction to researchers trying to determine a reasonable definition of an optimum at more than one z-score point. It would be interesting to research the change in the slope of the optima in relation to the span of the mortality risk curve. Steep ratios would indicate a very small range for optimal birth outcomes where deviations quickly increase mortality risk. Flatter ratios would indicate the opposite: infants have a larger "target" to be born into and still maintain at or near the lowest mortality risk for the population.

4.2 Traditional Model Comparisons

Of additional interest are the results comparing traditional approaches to the SPF model. It is quite clear that much methodological and statistical evidence exists to support the move toward more complex models of birthweight and gestational age effects on infant mortality and morbidity and beyond. When comparing traditional model parameter estimates with those of the SPF model as well as model fit statistics, it becomes clear that increasing both the degrees of freedom in the SPF model and the amount of information used increases the clarity of the risk picture. The utilization of eight birth outcome parameters versus three invariably provides increased power to quantitatively represent and describe the three-dimensional topographic infant mortality risk landscape. What this means is the ability to better represent the non-linear aspect of this multi-dimensional reality. This method allows for the use of continuous data after cut-points are developed for optima on both birthweight and gestational age. This component of this approach far outperforms the traditional, categorical approaches by allowing for various non-linear effects to be captured and modeled.

4.3. Race/Ethnic Considerations

4.3.1 Race/Ethnic Results and Comparisons

Moving toward more sociodemographic concerns, the race/ethnic pattern of results gives us much to ponder. Of special importance and a major impetus for

much work in this area are the race/ethnic differentials in infant mortality risk between White and Black populations in the U.S. Much research, debate and comment have provided an impetus to discover the many factors contributing to this continuing and widening risk (Bell et al. 2006; Frisbie et al. 2004; Grady 2006). Since birth outcomes are of major concern among researchers and public policy makers (Healthy People 2010; 2020), it is important to do as much as possible to answer basic questions about the relationship between these birth outcomes and infant mortality and morbidity. This dissertation continues the groundwork laid by SPF in experimenting with alternative modeling strategies in order to better elucidate the realty of this important relationship. With these ideas in mind, the results for the race/ethnic groups presented are important to discuss.

With the White population serving as a reference, we now turn our attention to Black and Mexican American infants. The results for Black infants in particular are of special interest as they are an important demographic minority in the U.S. with a very high infant mortality rate. With a rate differential hovering around two as compared to both Whites and Mexican Americans, the models presented attempted to shed additional light on this differential. With the standardization of each race/ethnic groups birthweight and gestational age distribution in effect, it is somewhat surprising to witness virtually stable ORs for Black infants in SPF models and *increasing* odds in GAM-calculated optima models after controls for birth outcomes are added. Period two does reverse this

trend among the GAM set of models by showing modest decreases in the Black OR as birth outcome controls are added. Although others have confirmed this finding for period two (Powers et al. 2006), the reasons are less clearly understood for the over-time variation. Even though bivariate representations of both birthweight and gestational age effects on infant mortality show that, standardized, Black infants exhibit increased risks across the spectrum of both distributions, these multivariate models do not reduce the differential when controlling for these birth outcomes and their interactions simultaneously. Given the issue of optima discussed above, the large change in optima for Mexican Americans may have unduly influenced the full models where race/ethnicity is a covariate. Moving to race/ethnic-specific models, there appears to be confirming evidence of the Black advantage in age-specific birthweight risks on either side of an optimum.

Turning to Mexican American infants, the most direct result from all models run is the basic identity in pregnancy outcomes that they exhibit relative to Whites. The Epidemiologic Paradox is clearly observed. The only departure from this result occurs in period two, Model 2 of Table 14. With an OR of 1.49, there seems to have been some indication of increased risk as the model included controls for birth outcomes. This interpretation is tentative at best due to the aforementioned change in the optimum over time for this group. Relative to Whites, Mexican American infants went from an optimum that was above Whites to one well below over time. This again is an important caveat in the

interpretation of this result. Given that Mexican American women have almost equal birth outcomes to those of White mothers, in spite of their lower socioeconomic status and poor rates of prenatal care use (Markides and Coreil 1986; Collins and Shay 1994; Cramer 1995; Albrecht et al. 1996; Singh and Yu 1996; Frisbie et al. 1998; Hummer et al. 1999; Landale et al. 1999), there has been much interest in explaining the origin of the epidemiological paradox as well as its significance. The leading explanation involves the positive migration selection hypothesis, which suggests that salutary behavioral practices from the country of origin, such as a healthy diet, infrequent use of cigarettes and alcohol during pregnancy, as well as positive family and social support systems, lead to positive pregnancy outcomes in spite of disadvantaged socioeconomic status and discrimination (Rumbaut and Weeks 1996; Guendelman 1998; Hummer et al. 1999; Landale et al. 1999a).

CHAPTER 5: CONCLUSIONS AND FUTURE DIRECTIONS

In summary, this study has served two main purposes: 1) a statistically assisted calculation of sex-specific, race/ethnic-specific and gestational age-specific birthweight optima over time, and 2) the incorporation of these new diverse optima into a sophisticated infant mortality-modeling scheme that begins with only birthweight and gestational age as the main independent variables of interest. These two aims served to produce insights into the form, function and relationships of these two important birth outcomes and their direct effect on infant mortality. The utility of this study should be measured not only on the results themselves, but also on the insights into complex phenomena and questions for future research it produces. It is my goal to continue to stimulate discussion in this area of research as those who have come before me have sparked my ambition to contribute, however minutely I may have done so. What remains to be focused upon are the important conclusions arrived at, contributions made and directions hinted at within this work.

The first major question that needs to be addressed is the following: How do these results impact race/ethnic research in this area? Complex statistical models will not lend themselves to easy interpretations and discussions. On the contrary, the reason for their complexity is the desire to model a process (maternal

health effects on pregnancy outcomes) that is *extremely* complex along a variety of dimensions: social, cultural, economic, biological, physiological, genetic and so forth. It has always been the aim of this line of inquiry to understand the pathways and processes discussed (birth outcomes) in order to statistically control for their effect on infant mortality and morbidity in the most correct way possible. The main impetus for this research was to clarify the role between birthweight and gestational age in order to correctly control for their independent and multiplicative effects, not to model them as outcomes or consider them as proxies for risk. The equation in question is defined by outcomes that are deleterious to infant health and well-being and inputs of interest are sociodemographic, socioeconomic, sociocultural and social psychological ones. As Hertz-Picciotto states well enough in her paper calling for abandonment of birthweight adjustment strategies in perinatal and infant mortality research: "There is at least one circumstance in which adjustment for birthweight would likely be justified. If one were investigating the impact of postnatal exposures on mortality, then adjustment for birthweight, which precedes such exposure, would be reasonable." (Hertz-Picciotto 2003:115). This is exactly the reason for research such as this. When adequate methods are used to control for an intermediate variable that precedes exposure, but is directly affected by factors of interest and also directly affects outcomes of interest, more adequate models need to be constructed in order to inform public and political discourse on issues relating to health

inequality within the U.S. For example, socioeconomic status affects both birth outcomes and infant and child mortality and morbidity independent of birth outcomes. Race/ethnicity continues to be a socially constructed risk factor with objectively important statistical, cultural and social outcomes.

What have we learned given the focus on race/ethnic health disparities as defined by differential infant mortality risks? We have learned some important lessons that will serve us well as we move forward within this line of research. The ability to accurately model birthweight optima by gestational age will allow for future research to track any changes in race/ethnic-specific optima as an end in and of itself. Although the reality of a moving optimum is not easily understood as of yet, the simple fact that different race/ethnic groups maintain different optima at different points in time is an important piece of this puzzle that must be further explored. Further analyses may uncover strong correlations between socioeconomic and/or sociodemographic changes and optimum changes. More research must focus on the biological and sociological etiology of fetal growth and maturity as a means of tracking population health levels. This means intense scrutiny of the birthweight link to infant mortality as well as scrutiny of birthweight itself as an outcome in regard to increasing or decreasing optima. As Wilcox himself states after suggesting that analyses that control for birthweight are unsound: “Even so, the association of birthweight with so diverse a spectrum of heath outcomes is a genuinely interesting phenomenon. Despite the thousands

of papers on birthweight published in past decades, there may be no subject in all of epidemiology more ready for creative—perhaps even revolutionary—insights." (2001:1240).

This study has also served notice of the importance of continued research and discussion of race/ethnic differences in birth outcomes and infant mortality. There would seem to be a clear lack of a simple, all-powerful, statistical solution to the Black/White gap in infant mortality, irrespective of our attempts so far to outline such a scenario. What research such as this has pointed out is that our sociological models are woefully inadequate for capturing the complexity at work within the human pregnancy process. What is required is a marriage of social and medical models in order to begin to link the social components of the prenatal sociological milieu to the physiological processes concerning *in utero* infant growth, development and birth. I think it necessary for those of us engaged in demographic endeavors to continue to push for this marriage of paradigms and continue to create and sustain research that pushes us and draws others to this goal. There exists a wide chasm between the sociological and biomedical research worlds, and it is research such as this that hopefully displays our limitations in this field. If Wilcox and others are correct, the ability to describe and explain the Black pregnancy differentials rests in epidemiological/biomedical explanations, not simple statistical correlations between measures of growth and disadvantaged outcomes. For if we truly care to disentangle the web of social factors that

disadvantage certain groups in society within health and development domains, we must strive to ask even more insightful questions before we put pen to paper (or fingers to keyboard). The work begun by many (cf Wilcox and Russell 1986), focused by Solis, Pullum and Frisbie (2000), and continued by myself and Powers et al. (2006) seek to illuminate on important demographic questions and develop preliminary models to describe the complexities inherent in physiological processes that are of such critical importance to the socioeconomic well-being of various groups in the U.S. and abroad. Work by Wilcox and colleagues also serves to ask these important questions within the epidemiological community. Both communities have at their core a strong desire to create paradigms that would allow society to directly address such large inequalities. It is my goal that the results presented in this dissertation continue to stoke the fires of inquiry into this important question and continue to improve our mission, a mission that has from its inception in sociology, focused primarily on reducing social inequality and improving our physical, social and emotional way of life.

APPENDIX

Table A1. GAM Logistic Regression of Infant Mortality by Race/Ethnicity: 1989-1991

	NH-White			NH-Black			Mex American		
Gestational Age	0	1	2	0	1	2	0	1	2
Early (E)	1.45	1.55	1.48	1.36	1.35	1.08	1.42	1.45	1.22
Late (L)	1.13	1.40	1.42	1.14	1.12	1.08*	1.11	1.05†	0.46
Birthweight									
Small (S)	0.91	1.13	1.10	1.58	1.69	1.31	1.09	1.22	1.10
Heavy (H)	0.31	1.25*	1.85	1.29	1.86	1.35	0.91†	1.53	1.52
E*S			1.01			1.08			1.06
E*H			0.93			1.13			0.96†
L*S			0.99†			1.01†			1.20
L*H			0.86†			1.02†			1.48
Intercept		-7.13	-6.99		-6.77	-6.07		-6.75	-6.39
-2LL		192,138	191,820		88,780	87,647		28,092	27,724

NOTE: Unless otherwise notes, all odds significant at 0.01 level; *p≤0.05; †p>0.05

Table A2. GAM Logistic Regression of Infant Mortality by Race/Ethnicity: 1995-1997

	NH-White			NH-Black			Mex American		
Gestational Age	0	1	2	0	1	2	0	1	2
Early (E)	1.45	1.51	1.35	1.36	1.44	1.34	1.43	1.42	1.36
Late (L)	1.14	1.24	1.06†	1.16	1.40	1.34	1.15	1.07*	0.93†
Birthweight									
Small (S)	0.92	1.10	1.05	0.87	1.12	1.08	1.25	1.14	1.07
Heavy (H)	0.22	1.41	1.31*	0.28	0.61	1.28†	0.95†	1.09†	1.02†
E*S			1.03			1.02			1.02
E*H			1.04†			0.97†			1.00†
L*S			1.03			1.00†			1.04*
L*H			1.01†			0.94†			1.35*
Intercept		-7.01	-6.75		-6.75	-6.54		-6.61	-6.48
-2LL		144994	144272		62817	62572		29550	29518

NOTE: Unless otherwise notes, all odds significant at 0.01 level; *p≤0.05; †p>0.05

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